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THE HYGIENE OF MEDICAL CASES, PARTICULARLY IN HOSPITAL WARDS:

WITH NOTES ON VARIOUS WARD INFECTIONS, AND OBSERVATIONS RELATING TO THE PROBABLE ROLE OF SECONDARY GASTRO-INTESTINAL INFECTIONS IN TYPHOID FEVER.[1]

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In the course of the last five years I have been much interested in watching for evidence of the transmission of infections of various kinds in medical wards; and during the latter half of this time I have been especially interested in the question of prevention, for I have become increasingly convinced that infections occur in medical wards much more commonly than I had suspected, and more commonly, I am sure, than the publicly and privately expressed views of physicians, so far as I am acquainted with them, would indicate.

In considering the circumstances in hospitals with which I am connected, and in others that I have visited, it has seemed to me that the system used in regard to the infectious diseases received in medical wards is usually much looser and less comprehensive than is that used in surgical wards; and that the customs in medical wards should be made to approach more closely to those used in surgical wards. In most of the governing principles, except in regard to the channel of entrance of the infection, the problem is much the same.

[1] Read in part at the meeting of the Association of American Physicians, Washington, D. C., May 7, 8, and 9, 1907.

It is entirely natural that, when once the beneficence of the aseptic care of surgical conditions had become apparent, there should have been a rapid and enthusiastic progress in improving the methods used for the protection of surgical cases; and that the purpose in view should have at once become the prevention of the conveyance of any infection to any patient, and particularly, in surgical wards, the prevention of the transmission of infection from any patient to another, so far as these purposes could be accomplished through the use of any reasonable measures. It is equally natural that there should have been a feeling that medical infections constitute an entirely different order of things, and cannot be approached in the same way; for different and much more complex circumstances are met with in dealing with them. It is likewise natural that much less attention should have been devoted to the prevention of medical infections, for they are essentially less important than the surgical, because less frequent; but the very fact that they are less frequent has led to an insufficient and a slower appreciation of their actual importance, and to their being less vigorously combated. The surgeon so regularly gets bad results, if his methods are bad, that it has been comparatively easy for him to see his errors and to correct them. The physician gets bad results so irregularly, or infections appear clinically so long after exposure, that their real importance and their sources do not readily become apparent, and bad methods may easily be considered satisfactory.

In recent years there has been such a rapid accumulation of testimony regarding the importance of direct transmission of all sorts of infectious diseases from human being to human being by direct or almost direct conveyance, and there has also been such a mass of evidence of the importance, as carriers of disease, of those who come into contact with the sick and afterward convey the causative agents, even though they do not fall ill themselves, that one may justly suspect that in hospital wards, where numerous persons of reduced resistance, many of them suffering from some infection, are brought together, and where those who care for them go from infected to uninfected, there may be much more frequent transmission of infections than we have believed to be the case. There is great difference of opinion as to the actual frequency of such occurrences, and I may have come to consider them more common than they are.

I do not believe, however, that anyone who looks into the matter in the light of modern studies of the transmission of infections will fail to find that what must, for safety's sake, be considered transmission occurs with sufficient frequency to convince him of its serious importance and of the great desirability of making more comprehensive provision against it than is now done.

To this latter statement I think all those who have already interested themselves in this question will agree. What I have to

say refers chiefly to those infections that have until recently been classed as non-contagious or, at most, very slightly contagious. Highly contagious diseases are, of course, subjected to very strict rules. For many years also there have been routine regulations in all well-conducted medical wards regarding some of the most important of the other specific infections. These regulations have especially related to tuberculosis and typhoid fever; to a less extent, to pneumonia; still less, to some other common infections; to some of them, not at all.

Faults in Customary Methods. The regulations ordinarily used may, however, reasonably be considered to be inadequate in at least two ways: First, they refer to only a few of the infections that are frequently received in medical wards; not to all of them. This, again, is quite natural, for diseases of this group were once thought to be rarely, if ever, transmitted directly. We have learned only one by one that many of these may be transmitted; and, as this was demonstrated with the most important diseases, regulations were applied to them. At the present time, however, we may safely say that any infection may be transmitted in a hospital ward if conditions favoring its transmission are present; and I think that anyone who observes the conditions in hospital wards must admit that, for most of these common infections, conditions that at least render their transmission more or less readily possible are often present. Considering the knowledge that we now have, it appears to me that the purpose of regulations in medical wards should be the same as in surgical wards; they should aim to prevent transmission of all infections, so far as this is feasible. This is a much more difficult undertaking than is the proper management of surgical wards, and is much less likely to be generally successful; but it is not to be shrunk from if it is a reasonable proposition.

The second objection to the customary regulations is that they cover only a portion of the means of conveyance, even with those diseases in regard to which especial measures are used; and the means of conveyance that are covered are not always those that are the most important sources of infection to the other patients in the wards.

For example, there are rules regarding the disinfection of the excreta, the bedclothes, and the food utensils of typhoid patients; but there are usually at best only indifferent regulations regarding the hands of the nurses, internes, and visiting physicians who come into contact with both typhoid patients and others. Moreover, the regulations that exist regarding the hands are, as a rule, looked upon as being established chiefly for the protection of the doctors and nurses themselves, rather than of the patients; and they are, therefore, often considered to be more or less voluntary. The excreta, however, even if disposed of without disinfection, are more dangerous to the outside public than to the patients in the wards;

the bedclothes are more dangerous to those in the laundry; and the food utensils, while important, would constitute but a limited source of infection if they were cleansed without actual disinfection; but the hands of the ward staff, especially the nurses, are frequently in contact with the persons, the bedclothes, and the excreta of typhoid patients; and subsequently these same hands are often used to examine or treat the mouths of other patients and to prepare their food. One of the most probable sources of ward infections with typhoid fever is, therefore, ordinarily controlled in a very indifferent manner.

In addition to the reasons that I have given, the regulations in medical wards are very frequently inadequate, because no one in authority knows whether they are carried out or not; and, frequently, neither the visiting staff nor the interne knows exactly what the regulations are or how they are intended to be carried out. Not uncommonly, they are devised by the chief nurse, with at most no more than a hasty approval by the medical staff. This latter point constitutes, perhaps, the most striking evidence that surgeons and physicians look at ward infections from different points of view. The surgeon's system is his chief interest and care. With physicians, however, the system is in frequent instances a matter of comparatively minor interest; and indeed many physicians regard it as being of such slight importance that they do not even know the details of the system used in their wards.

A year ago I had a conversation with a woman whose experience in the conduct of hospitals is unexcelled, in regard to numerous details that have to do with what I may call the aseptic care of medical patients. At the end of the conversation she told me that she had often discussed such matters with surgeons, but had never before done so with a physician. I would at once disclaim any appearance of vanity in mentioning this; I would, on the contrary, adduce it as very direct evidence that, with many others, I had not taken advantage of years of opportunity to go into these matters.

In recent years there has been a much more general interest among physicians than there was before regarding ward infections, as we have more clearly learned the possibility of their occurrence and the frequency with which they can be determined to occur, and some of the details of the hygiene of medical wards have been much improved. It is but just to state that if the matter is inquired into it will be found that the improvements have in many instances been largely due to the initiative of the chief nurses. Physicians have contributed some valuable details, but ordinarily they have offered as the most marked means of advance the more extensive use of isolation; and this measure has been somewhat generally adopted in dealing with pulmonary tuberculosis, and is coming into favor, though not yet frequently used, with typhoid fever.

Isolation an Incomplete Solution. I have the utmost sympathy with the use of isolation in the broadest and most intelligent way possible, and I shall mention the manner in which I constantly attempt to make the best use of it that I can with the means at my command. I feel, however, at the same time, that there is an erroneous tendency to believe that isolation is the most complete and ideal scheme that we can adopt. The use of it largely reduces the chances that those diseases that are isolated will be conveyed to other patients, but it alone will not protect the nurses; and, as I shall indicate, this should be an important part of the system in hospitals. Furthermore, it is a measure that can be applied to very few diseases; and I believe that our first object should be to attempt to devise a system that would reduce the possibilities of the transmission of all infections. In addition to this, even if isolation were possible with all the most important diseases, we should, in carrying it out, collect together groups of patients that, while having the same clinical diagnosis, would by no means be of indifferent influence upon one another. Many of the common infections are frequently complicated by secondary infections; indeed, some of them are in the majority of instances. I believe, for reasons that I shall give, that this is frequently true of typhoid fever, in addition to those diseases in which it is commonly accepted as of importance; and when we consider pneumonia, "influenzal" attacks, tonsillitis, and some other more or less serious common infections, we find that under any of these clinical diagnoses we should, in carrying out isolation, actually be grouping together a number of conditions that bacteriologically are often entirely different diseases. Isolation would, therefore, protect those who are not isolated from those who are; but, unless more detailed precautions were taken, it would not protect those who are grouped together in isolation from acquiring infections from one another. Isolation, then, to my mind, is an extremely important part of a complete system, but it is only a part, and there are numerous details that are not covered by isolation, but that need supervision and regulation.

In order to indicate my reasons for adopting the methods that I now use in my wards, and at the same time to illustrate the character of the regulations that I think desirable, I shall describe briefly some of the experiences that I have had with ward infections. Part of these have already been reported. Most of the observations that I shall especially refer to were made at the Episcopal Hospital, where we have constantly had a large number of cases of infectious diseases in the wards. The conditions there were, as a consequence, peculiarly favorable to observations of this character. The number of instances of transmission of infections that I have been able to observe there is, of course, larger than would be the case in a similar period of time in a hospital in which few such cases are received; but large groups of cases are necessary in order to make evident condi-

tions that would not appear if smaller numbers were under observation. In other ways the conditions in the Episcopal Hospital provide an entirely just comparison with well-conducted hospitals in general; for, in regard to nursing and other circumstances, everything has been quite as satisfactory there as in any of the hospitals with which I am acquainted. Indeed, almost all conditions have been exceptionally satisfactory, beyond the fact that the nurses have, at times, been overworked when unusually large numbers of seriously ill patients were in the wards. I do not think that the infections that I have seen could be attributed to inefficiency or carelessness. On the contrary, they were due, I believe, to errors in the methods customarily used in such wards, and the correction of these errors lies in the power of the medical staff of hospitals, and not with the nurses. I have seen more conditions of this kind recently than I did previously, and more than are usually considered to occur, simply, I believe, because I have carefully and deliberately looked for them recently, not because the conditions were exceptional during this time.

While studying the frequency and importance of ward infections, I have devoted some attention to observing numerous details of the care of patients in various hospitals, and have found it exceedingly easy to discover many ways of greater or less importance in which infections could readily be carried. Indeed, in a few instances, I have found customs that so completely disregarded the most evident opportunities for carrying infection that it would seem that there is sometimes a feeling that medical infections are presided over by a different and more beneficent providence than are the surgical. I have found, for example, in a ward in a well-known hospital, that the duties assigned to one nurse were to disinfect the bedclothes and the excreta, and to prepare the liquid diets.

There are very many ways in which infections may be carried; and, since many different infections are met with, it will be impossible for me to do more than to illustrate the things that seem to me to demand attention first of all, because they constitute the most serious dangers, and to indicate some of the ways in which the danger may be reduced without an unreasonable amount of labor and expense. By giving continued attention to the details of the management of these infections, we can, undoubtedly, learn readily many more things that should be done. The suggestions that I have to make I do not by any means consider perfect or complete. They are in part my own; in part they are used in some other hospitals. Certain types of infections have especially interested me; and they will serve to indicate the chief dangers in medical wards, and also the direction that I feel we should take in attempting to eliminate the most important sources of infection.

The whole matter may most readily be approached by first considering the sources. It is apparent that infections are most likely

to occur through the food; through the hands of the doctors and nurses, or other attendants upon the patients; through instruments and food vessels, or other ward utensils, such as bedpans and urinals; or through the atmosphere. There are, I think, a number of very evident ways in which protection against these sources of infection is not provided for by the methods now customarily used, even in the best hospitals.

Infection of the Food. So far as concerns infections, the most important of all foods in hospitals is, of course, milk; and, while much has been written about milk in recent years and some improvement has been made, conditions have actually improved so little that they still, in most institutions, need very earnest attention. In the first place, as to the condition of the milk when received. In hospitals that treat such patients the milk supply for babies and young children has, in many instances, been much improved, though there are still many hospitals that provide for young children milk whose source and hygienic condition are absolutely unknown. The most serious dangers are usually overcome by pasteurization. While this measure has its drawbacks and dangers, it is such a tremendous advance over the use of bad raw milk that it is fairly satisfactory; but it is not comparable to the use in the beginning of a reliable milk, for it is about on a par with simply subjecting soiled dressings from infected surgical cases to a high temperature and then using them with uninfected cases.

With medical cases in more mature subjects the conditions in regard to the milk supply are vastly less satisfactory, and much less has been done to improve them. The conditions in hospitals may, I think, be fairly represented by the following statement: A few hospitals have a supply that is actually known to be good, because it is under the supervision of a medical milk commission, or is regularly supervised by representatives of the hospital, from its source to its delivery. A few have a supply that is complacently called their own, or that is under their own control, but is really very inadequately supervised; and, when actually investigated, the milk is found to be very little, if at all, better than ordinary market milk. The great majority of hospitals, however, obtain their supply simply by contract with a dealer, and they usually know nothing about its sanitary condition when given to the patients, although in a certain number of hospitals more or less frequent bacterial counts are made. If these counts are, as a rule, fairly satisfactory, the milk is considered to be reliable. Unless it is an exceptionally good milk, the counts are frequently found to be temporarily very high, but these high counts are usually passed over as inevitable, and any bad results from the use of the milk are ordinarily not remarked upon. The improvement, therefore, in the character of the milk supply is actually slight; and only a small proportion of hospitals exercise any adequate control over the condition of the milk when it is

received, while very few adequately control the hygienic conditions at the dairies and in the course of transportation. These latter matters are much more important than mere bacterial counts. In the first place, a dealer can, by surreptitious pasteurization at a low temperature, keep the bacterial count of a bad milk down to a low point, without its being known that he has done this. While this makes the milk safer if it is well done, it is likely to make it less safe if it is badly done; and often it is badly done. Aside from this, bacterial counts alone are a very imperfect indication of the condition of the milk, for they, at best, indicate only that the total bulk of infection that has reached the milk is small; or, more commonly, they simply indicate that the milk has been kept well cooled. They do not at all satisfactorily indicate how dangerous the infections that are present may be, and they do not demonstrate whether the conditions at the dairy and in the course of transportation are such as to make it improbable that the bacteria producing typhoid fever, tuberculosis, diphtheria, and other specific diseases, as well as the much more common bacteria that produce more or less serious gastro-intestinal disturbances, could gain entrance to the milk in dangerous amounts and are likely to do so with dangerous frequency.

The only ways in which such evil possibilities can in the beginning be reduced to a minimum are, for hospitals: (1) To secure certified milk (and this is usually too expensive, and often impossible because such milk is not available in large quantities); (2) to secure their own dairies, or to have dairies supply them directly, and to have the condition of the milk on delivery controlled by regular temperature records and frequent bacterial counts, and the condition of the dairies controlled by sanitary and veterinary inspection; or (3) to have concerted action taken by medical milk commissions and hospitals, whereby a guaranteed milk that is reasonably sanitary may be supplied to hospitals at a moderately increased cost, the hygienic conditions surrounding its production and delivery being under the continuous supervision of the milk commissions. It is earnestly to be hoped that the latter proposition, which is under consideration by the Association of Medical Milk Commissions, may be adopted. It is the most reliable and the least expensive solution of the question now apparent. Many hospitals would soon be persuaded into acting with the commissions, while others that might be reluctant would be forced into doing so through the general enlightenment that would come from the partial introduction of such a measure. At present, however, this solution of the question is not available; and it will not be widely available for some time, for a number of reasons. One important reason is that, as anyone who has tried to find a suitable dairy for a hospital supply will know, there are now in the neighborhood of most cities very few farmers who could supply milk that would pretty constantly pass as sanitary. Hence

the farmers also will have to be led into the movement in large numbers, though this will quickly follow the demand.

I have given this very cursory statement of the situation regarding the milk supply in order to indicate the reasons why, as matters are at present, I have been reluctantly forced to the definite conclusion that, except in those few hospitals in which the supply used is carefully and continuously supervised, and is known to be entirely reliable, all milk that is given to patients that are seriously ill, and especially to patients whose gastro-intestinal tracts need careful management, should be pasteurized, whether the patients be adults or children. I have reached this conclusion reluctantly, because pasteurizing adds trouble in the wards and leads to constipation in the patients, and pasteurized milk often becomes somewhat repulsive to adult patients after a short time. Nevertheless, these are minor objections when compared with the bad results that raw milk may produce.

Dangerous Results from Infected Milk. Some years ago I reported widespread and serious abdominal symptoms in the typhoid patients at the Episcopal Hospital, due to the use of a supposedly good milk, which investigation proved to be actually very bad[2] The abdominal symptoms were reduced from alarming prominence and frequency to an unusual inconspicuousness by merely pasteurizing the milk. Subsequently, the milk supply was changed; and the best supply that appeared to be available, exclusive of the certified milks, which are too expensive to be reasonably used in a large general hospital, was secured by the managers, at an increased expense. Rigid regulations concerning its temperature and bacteriological condition upon delivery, and its care after delivery, were established, and pasteurization was stopped. All trouble with milk appeared to cease for some time; but during my next service, I twice noticed that on one day several typhoid patients developed sharp diarrhœa. Upon inquiry, directly after these occurrences, I learned that Dr. Ghriskey had each time found that the milk yielded high bacterial counts on these days. The counts at other times had run very low. These conditions were transitory; but in December, 1905, diarrhœa developed practically coincidently in a number of typhoid patients in the men's and women's wards; and Dr. Miller and Dr. Stevens, who were then on duty, had the milk pasteurized again, because they got reports for several days from Dr. Ghriskey that the bacterial counts were continually running high. Upon investigation, it was found that the refrigerating plant of the man that supplied the milk was out of order. This had evidently caused the trouble, for high counts continued until the plant was reconstructed. This last-mentioned incident appeared to have serious results, for, as will be shown in the table that I shall give later, there were an unusual number of

[2] New York Medical Journal, March 25 and April 1, 1905.

perforations among the typhoid cases at this time and shortly afterward and serious diarrhœa was, as I have stated, very prevalent.

These experiences make it clear that, in spite of the fact that our milk supply is better than that of most well-managed hospitals, we are at any time liable, and most hospitals are still more liable, to unexpected trouble, which may have very bad results; and this may be due to either the misfortunes or the carelessness of those that produce and deliver the milk. Indeed, everyone who has any knowledge of the milk problem knows that all but the most rigidly supervised milks not infrequently show transitory bad infections. Frequent bacterial counts will not prevent trouble. When such counts are high, they indicate that something is wrong; but this is of little importance except in avoiding subsequent trouble, for the final count is obtained only forty-eight hours after the milk arrives, or at best one knows only after twenty-four hours or thereabouts that a dangerous number of bacteria are present, and meanwhile the patients have taken the milk and the damage is done. Such occurrences can be reduced to a minimum, if hospitals and milk commissions can work in unison in controlling the general hygienic conditions surrounding the production and delivery of the milk. Otherwise, it can be done only at an expense that is impossible for most hospitals to undertake. In the absence of either of these safeguards, I believe that pasteurization is the only reliable measure.

The bad results are not confined to typhoid cases. I have notes of four pneumonia cases in which troublesome diarrhœa occurred at the same time that it occurred in the typhoid cases, in the several periods that I have mentioned, in which the milk was known to have been badly infected. I have likewise noted the same thing with patients of various other kinds, particularly with those whose gastro-intestinal tracts were already disturbed or had recently been disturbed.

It is to be especially observed that pasteurization must be properly carried out if it is to make the milk safe. This remark is not so superfluous as it may seem, particularly in managing large groups of cases, when the pasteurization must ordinarily be done in large bulk. I was recently told that the pasteurized milk that they were using in a ward that I visited appeared to be causing gastric and intestinal symptoms. I went over the various steps used there in the process, and finally took the temperature of the air in the compartment of an ice-chest in which the milk was being "cooled." Two hours after the milk had been placed there the thermometer registered 78° F. The ice-chest was entirely unequal to the task imposed upon it. The trouble was overcome by placing tightly covered vessels that contained the pasteurized milk in large receptacles and surrounding them with cracked ice; it was summer time, and the tap-water was too warm to be of service in cooling the milk. It should be known just what all the details of pasteurization are as

the process is carried out in each instance. Bacteriological studies by Pennington, Evans, Bergey, and others have sufficiently shown that poor so-called pasteurization may simply make bacteria flourish.

Care of the Milk after Delivery. The care of the milk does not cease, however, with the points that I have touched upon. Milk may be good when it gets to the hospital, but very bad when it gets to the patient, and some other foods, especially those that are liquid, may likewise become dangerous in the course of their preparation. In the first place, there should be a definite and suitable system of keeping the milk properly refrigerated after it has been received. I have seen milk, after delivery to a hospital ward, stand for an indefinite time in a warm corridor, because it was nobody's especial duty to look after it and nobody had had a good opportunity to do so.

The system that the managers have established at the Episcopal Hospital follows the proper principles very closely. The milk is received by a graduate nurse, who is required to make an immediate record of the temperature of the milk in each can, and then to have it placed at once in the central cooling-plant, where it is kept at a temperature that is regularly recorded and must be below 45° F. The cooling is done by means of freely iced water. The same nurse is responsible for the expeditious delivery of the milk to the various wards, in the amounts and at the times required. At the wards, it is delivered to an especially designated nurse, who is required to see that it is at once and subsequently kept properly cooled, and that it is protected from infection.

Precautions against Infecting Food in the Ward. The latter point is one that is disregarded with surprising frequency. It is, indeed, even more generally disregarded at present than is the condition of the milk on delivery. Everyone is alarmed at once by the knowledge that a person who is coming into daily contact with a case of typhoid fever is working in the dairy from which a milk supply comes; but there is certainly no common concern over the fact that in a very large proportion of hospital wards the general milk supply of the ward is being constantly drawn upon by nurses who at various times soil their hands to a more or less dangerous degree through caring for the mouths, the persons, the excretions, and the bedclothes of patients with typhoid fever, pneumonia, tuberculosis, and a variety of other infections. I have, indeed, mentioned an instance in which a nurse actually had assigned to her as her chief duties the preparation of liquid diets and the disinfection of excreta and bedclothes.

At the Episcopal Hospital we have for some years had one nurse assigned to the sole duty of preparing and delivering to the patients (but not administering) the liquid diets in the medical wards; and this is the ideal system. The enormous importance of infection of the food by nurses who care for the bodies and the various wants

of patients has been sufficiently demonstrated by the work of Heubner and other pediatrists, and these observers have, likewise, demonstrated the wonderfully gratifying results that may be obtained by eliminating this danger. The system mentioned has been feasible at the Episcopal Hospital, because we have had there such large numbers of acutely ill patients that the diet-nurse's time was entirely occupied in this way. In any ward, however, it is possible, and is important, that one nurse alone should handle the ward milk supply and any other general supply of food that is not to be cooked subsequently; and the duties of this nurse should not include the care of the persons, the excreta, or the bedclothes of any infectious case, or the handling of any objects that may have been contaminated by such cases.

Regulation of the Working Personnel of the Ward. Next to the care of the food, the point that appears to me to be of first importance is to establish a system that will reduce the chances that the working personnel of the ward—the doctors and nurses especially, and most particularly the nurses—will convey disease from one patient to another. It is, of course, probable that the mouth is the most important avenue through which infections, when conveyed by them, enter; and in treating patients who are seriously ill, the possibilities, with the system ordinarily used, of conveying infections into their mouths must arrest attention at once. A part of the nurse's duties in such cases is to cleanse the mouth of each patient under her care thoroughly and frequently, and in doing this she may easily carry infection into the mouth; for very commonly she is caring for the mouths, the persons, the bedclothes, and perhaps the excreta, of patients who have non-infectious conditions as well as of those who have infections. She, therefore, has a fruitful opportunity to infect those who are not already infected, and to communicate new infections to those who already have some form of infection. Even when caring for several patients who have the same disease, she may transmit secondary infections existing in one patient to others who previously did not have them. This point has particularly impressed me, both because of the evident opportunities for carrying infection in this way and because it has appeared to me to be the most probable source of some of the ward infections that I have seen.

Care of the Patients' Mouths. I have adopted the following system in endeavoring to eliminate trouble from this source:

If she is about to care for patients' mouths, the nurse, before beginning such work, sterilizes her hands, as they are considered to be constantly infected. Indeed, because satisfactory sterilization is difficult to accomplish and would often not be reached, I prefer that she should wear rubber gloves while preparing swabs for the mouth, and also while cleansing the patients' mouths. Before starting to attend to these patients, she prepares, at one time, a

sufficient number of applicators wrapped with cotton to make at least two for each patient whose mouth she is to treat. The applicators should be boiled before being used; they are best made of metal, and should be fairly heavy, so that they do not bend easily while in use. The most satisfactory scheme, when such a nurse is available, is to have the diet-nurse, or some other whose duties are such that she does not get her hands infected, wrap the applicators for all the nurses. In cleansing the mouths, the nurse uses one applicator to lift aside the lips, cheeks, etc., and the other, to cleanse with. She avoids touching the lips or the interior of the mouth, or, if she must do so, she at once cleanses her hands again afterward. The soiled applicators are put into a separate receptacle as they are used, and the next patient is then attended to, the whole group needing attention at one time being cared for consecutively. The nurse then removes the cotton from the applicators by means of hemostats, keeping her fingers away from the soiled cotton or metal. In this way, she can, with one sterilization, get her hands clean, and avoid soiling them afterward, while repeated sterilizations between patients would be practically impossible. In case she has at any time to cleanse individual mouths or to make applications to a patient's mouth or throat, her hands should always be sterilized before she begins to wrap the swabs or to treat the mouth, and her fingers should always be kept out of the mouth if possible.

When infectious diseases are present in the wards, and this is nearly always, the same care in treatment as that given an uninfected wound should regularly be given the mouths of the patients. This is often not done. Indeed, in a prominent recent book on nursing, the method of wrapping the finger with gauze that is wet with a mouth wash, and then swabbing the mouth with the finger thus wrapped, is still recommended as the best means of cleansing the mouth. This method is effectual, but is probably, from the standpoint of transmitting infection, the most dangerous that can be used, and I had supposed that it had already been finally condemned.

Visiting physicians and internes have not always a due respect for the mouth as an avenue of infection. I can well remember having examined mouths with entire forgetfulness of the fact that I had shortly before seen patients that had typhoid fever, tonsillitis, or pneumonia, for example, and I still frequently see this done. The hands are sometimes washed, sometimes they are not. When they are washed, it is often done more thoroughly after the examination than before, and it would appear, therefore, to be frequently done from a spirit more of estheticism than of asepsis. I now make it a special point to sterilize my hands before and after examining any patient's mouth or throat, if I must bring my hands into contact with the mouth. I would note incidentally that I also regularly wear a gown in making my rounds, partly because

of its moral effect, but much more because I wish to protect my clothing from infection.

Care of Ward Utensils. The measures that I have just suggested will aid in reducing the extent to which the nurses' hands come into contact with infection. Efforts of other kinds should be made to accomplish the same purpose for the protection of both the nurses and the patients. One prominent way in which this may be done is by boiling all ward utensils frequently, and especially directly after they have been used with infectious cases. Bedpans and urinals, in particular, must constitute a frequent source of trouble, as they are ordinarily cared for. Their contents are disinfected and, to some extent, their interiors, but with the interiors the disinfection is usually only imperfect, and the exteriors are scarcely disinfected at all. Bedpans and similar utensils must carry, much of the time, an extremely varied flora, and frequently a dangerous one. Immersion for some time after use in a disinfecting solution is a fairly satisfactory method, but it is more troublesome and less certain than boiling them. For this purpose, I know of no apparatus so simple and requiring so little trouble as that devised by Dr. Charles P. Noble and for some time used by him at the Kensington Hospital for Women. It consists of a box-like metal receptacle, large enough to contain a number of bedpans, with a faucet for the inflow of hot water, an outlet connected with a drain pipe, and a steam coil in the bottom, the latter being connected with the hospital steam pipes. Hot water can be brought to the boiling point in this within a few minutes, and bedpans and other dirty ward utensils, when made of agateware, can with the greatest readiness be boiled in it as desired, with almost no expense after it has been installed. It takes up very little space.

When thus made bacteriologically clean directly after they have been used, such objects, if handled carefully in emptying them, should be very slight sources of infection for the nurses' hands. In addition to this, although I have not yet insisted upon it, I feel that it is wise to have nurses who are in attendance upon patients with typhoid fever, infectious gastro-enteritis, dysentery, and such disorders, wear rubber gloves (these should, of course, be kept for this purpose alone) in cleansing the persons of these patients, in changing the bedclothes, at any rate if the latter are soiled with discharges, and in bathing the patients. This would eliminate most of the ready opportunities for soiling their hands with infectious material.

There would still remain as a conspicuous source of infection of the hands the necessity for rubbing the patients, and this cannot be done comfortably and well except with the bare hand. It would, however, be comparatively easy for nurses to sterilize their hands regularly after rubbing their patients, as this would require only infrequent sterilization, whereas it is impossible for them to sterilize

their hands after each bit of treatment of any sort that they give to the patients. If, therefore, they were protected against all the important sources of infection except that which comes from rubbing the patients, it would be feasible to eliminate this source also pretty thoroughly.

Orderlies should be eliminated from the direct care of patients as much as possible. The administration of enemas and similar forms of treatment should be carried out by the nurse. This is the only means by which danger from orderlies can be prevented, so long as it is so generally customary, as it is at present, to employ cheap laborers, who are often degraded persons, in this capacity. Offering higher wages would improve their quality, but it is questionable whether even then men could be secured who could be trusted with any real care of the sick, for the work is essentially repulsive, and the condition of subordination to everyone else in the ward would be too irksome to most persons of intelligence.

Many physicians will think all these regulations extremely burdensome and a good deal of a tempest over small matters, particularly when so distinguished an authority as Curschmann belittles the danger of ward infection with the most important of the diseases that come especially under these regulations—namely, typhoid fever. They are, however, not actually so difficult to carry out as they sound, and, as to the danger, a vast array of literature in recent years has demonstrated beyond all question the frequency of contact infection with typhoid fever, evidence in most massive form having been given by the report of Reed, Vaughan, and Shakespeare in their report upon Typhoid Fever in the Spanish-American War.

Danger of Typhoid Infection of Nurses. As a direct way of determining the importance of the questions under discussion to nurses and patients in medical wards, let us first consider the effects upon the nurses themselves. This will also serve as an index of the extent to which they may become carriers of infection to patients. In the four years 1903 to 1906 inclusive, we had 14 cases of typhoid fever in pupil-nurses at the Episcopal Hospital. The average number of pupil-nurses in the hospital during this period fluctuated closely around 75. There was, therefore, an average of 3.5 cases a year, and the morbidity among the pupil-nurses was, therefore, 4.66 per cent. During the same period, there were reported in Philadelphia 31,467 cases of typhoid fever—an average per annum of 7877 cases. The population of the city during this time was about 1,500,000. The morbidity of this disease among the citizens in general was, therefore, about 0.524 per cent. Consequently, the morbidity among the nurses at the Episcopal Hospital was 8.9 times greater than among the citizens in general.

At the Pennsylvania Hospital, they had, according to figures kindly furnished me by Dr. Scott, 18 cases among the nurses in nine years, or an average of 2 a year. During each year, they had

60 pupil-nurses in the hospital. Therefore, the average morbidity among the nurses was 3.3 per cent., or 6.3 times the morbidity of the average of the citizens of this city.

In Johns Hopkins Hospital (according to figures kindly given me by Dr. Cole from Dr. McCrae's statistics, which have since been published), they had 15 cases in their total series, that is, in a period of fifteen years, or 1 case a year. Dr. Cole could not give me the average number of pupil-nurses, but since the total of all nurses now ranges from 110 to 120, it appears proper to put the average number of pupil-nurses during this time at about 100. This would give a morbidity among the nurses of 1 per cent. The health reports of the city of Baltimore, kindly furnished me by Dr. Bosley, the Health Commissioner, show, during the ten years from 1896 to 1905, inclusive, 7294 cases of typhoid fever, or 729 per annum. The average population of the city during this period was 528,954. Hence the morbidity there among the citizens in general averaged 0.13 per cent. Consequently, the morbidity among the nurses at Johns Hopkins Hospital was 7.7 times greater than among the outside citizens.

Parenthetically, I would say that, while these figures are striking enough in all three instances, they appear to arraign the conditions at the Episcopal Hospital more severely than those at the two other hospitals, but this is due to the fact that we have had so large a number of cases of typhoid fever in the Episcopal Hospital. During the period that we had 14 nurses ill with the disease (that is, in four years), we had 3230 cases of typhoid fever in the hospital; that is, 1 nurse sickened to every 230 cases received. During the nine years in which there were 18 cases at the Pennsylvania Hospital among the nurses, 4467 patients with typhoid fever were received there, or 1 nurse sickened to every 247 cases. During the time that 15 nurses sickened at Johns Hopkins, there were 1500 cases, or 1 nurse to every 100 cases. Hence a proper comparison indicates that the conditions, while unfortunate enough, were not *relatively* bad at the Episcopal Hospital. They are, however, sufficiently disquieting anywhere, when looked at in the way that I have indicated, and this is, I think, the only proper way of viewing them. The mere absolute number of cases in nurses is not at all a direct indication of the real danger of infection, for the absolute number of nurses anywhere is small. The conditions inside a hospital must be compared with those outside in order to gain a proper conception of the actual dangers.

It may fairly be said that even the figures that I have given by no means represent the danger to those nurses who are chiefly exposed (namely, those in the medical wards), for these figures refer to the total number of pupil-nurses in each of the hospitals. If the morbidity were determined among the nurses engaged in medical wards, it would undoubtedly be much higher. I know of

the figures for the nurses working in the medical wards at Johns Hopkins only. Eleven of their 15 cases among nurses occurred among those thus engaged. If it be considered that one-third the nurses are engaged in medical wards (and probably there are usually not more than this), the figures then would be 0.73 cases per annum among 33 nurses, or a morbidity of 2.2 per cent., that is, 17 times the average morbidity of Baltimore citizens in general. The figures become still more impressive when one considers that nurses in hospitals, as well as patients, are much better protected from water-infection than are the outside citizens in general. Pupil-nurses are, of course, in that period of life in which typhoid fever especially occurs, and this is of some importance in explaining their high morbidity. It is, however, far from being a sufficient explanation of these figures.

Danger of Typhoid Infection of Patients. Nurses are then evidently to a most unusual degree, liable to contract typhoid fever. It must be granted that the liability comes chiefly through infection of the hands, and it is impossible to escape the conclusion that they may convey the disease, not only to themselves, but to others. However, the danger to others is less than to themselves because the infection becomes somewhat diluted in transit, and still more because nurses always have opportunity to infect their own mouths, while they have occasion to treat the mouths of only a portion of the patients that they are caring for. I would by no means indicate that I believe that all patients that acquire typhoid fever in hospitals get it through the medium of the nurses. There are a number of other important ways in which they may acquire it, and especially by helping about the ward when they are on their feet, but for those that acquire the disease when they are fast in bed, I think that this is an important source of infection. I have notes of 5 cases of ward infection that I saw in patients at the Episcopal Hospital in the period from 1903 to 1906, inclusive. Three of these occurred in persons that had been confined to bed. Their food was practically beyond suspicion as a source of disease, for it was cared for after the manner that I have described. It seems to me that the next most probable source was the treatment of their mouths. They had all been cared for by nurses who were at the same time giving attention to cases of typhoid fever.

It may, nevertheless, be objected that while it is apparent that the points that I have described as desirable regulations are of importance to the nurses, the number of infections that are known to occur in patients is, at most, too small to justify any very burdensome measures of prevention. Known figures, when properly looked at, do not bear this out, however. As I have said, I saw 5 cases in patients in four years at the Episcopal Hospital. My service there yearly is one-sixth of the total medical service, since six physicians are on duty each year. Owing, however, to the time of year that I

serve, I see somewhat more than my share of cases of typhoid fever. While I have not the figures for the experience of my colleagues, I feel that it is safe to consider that, all told, as many as 15 patients acquired typhoid fever in the medical wards in these four years. The number of cases in the medical wards averages about 125. Considering the average number of cases of yearly ward infections in patients to have been 3.75, this makes a morbidity of 3 per cent., or 5.7 times the morbidity of the citizens of Philadelphia in general. In Johns Hopkins Hospital, they have less striking figures, having had but 5 cases in the medical wards in fifteen years, and probably no hospital has a better record than this. These small figures do not, however, actually indicate the degree of danger, for with usually about 100 cases in the medical wards, they have a typhoid morbidity among their medical cases of 0.33 per cent., which is about 2.5 times the morbidity of Baltimore citizens in general. As I have already indicated, patients in hospitals are very much better protected against water infection than are persons outside. These figures also are, therefore, for this reason really larger than they seem; and in addition to this, a few cases probably develop after the patients leave the hospitals in which they acquire the disease, and these would somewhat enlarge the figures. The higher morbidity at the Episcopal Hospital is, again, due not to laxity in methods but to the larger number of opportunities for infection there. We have averaged over 800 cases a year in the period of which I am speaking, while at Johns Hopkins they have averaged 100. Putting aside, however, any question of relative morbidity inside and outside hospitals, I think that there is no doubt that any surgeon who for a series of years had had a yearly average of 3.75 instances of serious septicemias among patients and a similar number among nurses, due to conditions in his wards, would take very active measures to prevent other similar occurrences. The number of cases of typhoid fever in hospitals varies, of course, greatly, but the danger from each case in any hospital is, I think, fairly indicated by the figures that I have given for two representative hospitals.

I may be asked, Would not isolation of the typhoid cases give a much simpler and more certain solution of the question than the details suggested? This would largely protect the other patients from typhoid fever, but it would not protect the nurses in attendance upon the typhoid cases, and this is evidently a matter of immediate consequence. Furthermore, the problem is not one that relates to typhoid fever alone. There is certainly some danger that pneumonia, tonsillitis, and a number of other diseases may be carried in this way, and there is especial danger that various more or less nondescript gastro-intestinal infections may be so transmitted.

I shall mention some instances of the transmission of pneumonia later, and I have some reason for thinking that in a portion of these cases the infection was carried in this manner, but the question of

gastro-intestinal infections has especially interested me in this connection.

Secondary Gastro-intestinal Infections in Typhoid Fever Patients. During several years past I have, particularly, become more definitely convinced that secondary gastro-intestinal infections play an important role in typhoid fever, and I strongly suspect that they are at times introduced in the manner that I have indicated. If I am right in this view, the prevention of such occurrences is a matter of lively interest to the typhoid patients themselves, and if the latter were simply isolated from other patients and no other precautions were taken, they would be in danger of acquiring from one another more or less serious secondary gastro-intestinal infections.

The observations that have led me to this latter viewpoint are purely clinical, and are subject to correction as the result of bacteriological investigation. They are, however, so largely supported by the closely analogous conditions that are known to occur frequently in children's hospitals, especially in infant wards, and they appear to me so suggestive in themselves, that I have little hesitation in describing them briefly and in pointing out rather radical suggestions that must occur to one as a consequence. The train of observations was as follows:

The effects of infected milk in the various instances that I have described and the especially bad results in typhoid patients demonstrated convincingly what is *a priori* to be anticipated: namely, that typhoid patients are especially susceptible to secondary gastro-intestinal infections, and if they acquire such secondary infections the abdominal symptoms become much worse and the disease is, of course, made more dangerous. It is clear, therefore, that if occasion is given for them to enter, secondary infections may play an important role in the disease. In the epidemic of milk infections among typhoid cases that I reported, the effect of pasteurizing the milk was, as I noted at that time, such as to change the picture in the wards from one in which abdominal symptoms were alarmingly conspicuous to one in which they were reduced not only to normal frequency but to so inconspicuous a role that physicians visiting the wards with me remarked upon it as being unusual. With from twenty-five to seventy-five cases of typhoid fever in my wards continuously during my services since that time, I have had many periods, lasting for even weeks at a time, when not a single typhoid patient exhibited hemorrhage, perforation, noteworthy distention, or even diarrhœa that was at all severe. The same thing has occurred with my colleagues.

Outbreaks of Diarrhœa, Hemorrhage, and Perforation. These intervals were broken by periods, which were sometimes prolonged, in which we had constantly a number of cases of more or less severe diarrhœa and distention, and in which hemorrhage

and perforation occurred repeatedly, and sometimes in very quick succession in a number of cases. Everyone who has seen large numbers of cases of typhoid fever together has observed such waves of alteration in the character of the group of cases, and I had seen it before, but since the milk has been kept under careful supervision, waves of bad symptoms have excited my attention more sharply, because the periods of freedom have been more striking and more prolonged and have therefore given a better contrast.

These changes are usually attributed to the fact that milder cases are being received at one time, more severe at another. No doubt, this does explain part of the circumstances, but as an explanation for the whole, it is merely begging the question, at best, and it is difficult to understand why the character of a simple typhoid infection should vary so quickly and so frequently in the same community. Furthermore, I especially noted that by no means all the cases that developed bad symptoms were of unfavorable character when they came in, but that, on the contrary, many of them first showed these symptoms when they had been in the hospital for some time. I was also struck by the fact that the change in the character of the cases several times occurred very abruptly. In these instances, in a week after the conditions had been so quiet that no anxiety was felt over any except one or two very toxic cases, we would have a group of cases with very marked abdominal symptoms; and in repeated instances there was a quick succession of cases of hemorrhage or perforation.

Two incidents that I saw, within a few weeks of each other, especially impressed me as pointing to an explanation. In the first, a man, aged forty-four years, of robust frame, was admitted on the tenth day of the disease. He was but mildly ill, had been moderately constipated throughout, and had no special symptoms that aroused the slightest anxiety. An enema was given him thirty-six hours after admission, and he had a formed movement afterward, but a few hours later he developed an offensive diarrhœa with tenesmus, and rapidly became distended. These symptoms continued, and on the next day he had a very severe hemorrhage. He died twenty-four hours later, after repeated hemorrhages, his abdomen having meanwhile become enormously distended. In the bed next to him was a man who before admission already had had an offensive diarrhœa and marked distention. Upon inquiry, I found that the same orderly was in attendance upon both, that the orderly had given the first-mentioned patient an enema; that the rectal catheter through which it had been given had not been boiled before it was used; and that the orderly, as is usually the case with these employés, had not cleansed his hands before administering the enema. The nurses were overworked at this time, and it was necessary for the orderlies to give some of the enemas. Strict orders were, however,

given after this as to cleanliness, and the nurses were instructed to boil the catheters themselves before they were used.

Six weeks later another man, who had been in the hospital ten days and had been continuously constipated, developed a sharp offensive diarrhœa within a few hours after having received an enema. The diarrhœa was severe for about a week, but had no more distressing results than to make him more seriously ill than he would otherwise have been. I inquired into this incident also, and, to my chagrin, I learned that exactly the same thing had occurred as in the previous case, owing to the illness of the head nurse of the ward and the confusion resulting from this.

These occurrences made me feel increasingly justified in suspecting that typhoid cases may acquire secondary gastro-intestinal infections from sources other than food. The suspicion was further increased by noting that the stools of those patients who developed diarrhœa at about the same time were, in several outbreaks of diarrhœa, of exactly the same gross character, and quite different from what they had been previously, as well as different from the stools that occur in such patients as the result of what appears to be simple difficulty in digestion, namely their stools were, in some of the outbreaks, deep brown, in others green, and they were strikingly offensive.

Apparent Transference of Abdominal Complications. In my services during 1905 and 1906 I devoted especial attention to the sequence of events that occurred when abdominal symptoms appeared in a series of cases. The incidents that I found as a consequence, together with a study of the chronological sequence of hemorrhage and perforation in our wards during these and the two preceding years, have led me to the definite conviction that there is sufficient evidence that special causes of abdominal symptoms may be passed from case to case in typhoid fever to justify us in taking measures to prevent this. There is, likewise, evidence enough to make it very desirable that the bacteriological side of the matter be worked out. The latter has, of course, very great technical difficulties associated with it. Dr. Joseph Evans kindly undertook bacteriological studies of the stools in several cases that developed diarrhœa in two of the periods that I shall describe, and contrasted them with stools from patients without diarrhœa that were in the ward at the same time. In the first instance, however, he was, owing to temporary illness, unable to finish the study; and in the second the results were inconclusive.

Clinically, the sequences were as follows: On April 1, 1905, when I went on duty in the men's wards, I found that of 34 patients who were in the active stage of typhoid fever, 12 had decided diarrhœa, and 5 of them had considerable distention. Within a week, perforation occurred in 5 of the 12 patients with diarrhœa, and 1 had a hemorrhage. Only 1 other perforation had occurred since January

1, of the same year, and there had been but 1 hemorrhage in that time. I looked about for the cause of this and, of course, suspected the milk first, because of my previous experiences; but the milk was in excellent condition at that time. On looking further, I found that the records showed that 3 of the 12 patients had come in with offensive diarrhæa; 2 of these 3 had perforated. The other 9 had developed their diarrhœa one after another within ten days after these three had been admitted.

By April 10 the diarrhœa had subsided, and no patients with typhoid fever in the wards were dangerously ill. No further disquieting abdominal symptoms occurred until April 21, when 2 patients were admitted that had severe, offensive diarrhœa. On April 23, one of these men had a severe hemorrhage; and subsequent repeated hemorrhages caused his death two days later. Meanwhile, beginning April 22 and in the period up to April 29, 8 patients that had previously been free of diarrhœa developed it in some degree—from three to ten movements a day, in the various cases; and of these 8, 6 had severe hemorrhages in the period between May 2 and May 10. Three of these latter 6 died of hemorrhage, and a fourth of perforation.

By May 15 everything had become quiet again among the typhoid cases, and, except for several patients who were very toxic and a number who had slight diarrhœa with curds in their stools, but whose trouble quickly subsided after correcting their diet, there was no trouble up to June 9, except an isolated perforation which occurred, June 8, in a man who had not had diarrhœa. From June 9 to June 11, however, 3 patients were admitted who had decided and persistent diarrhœa, and in 1 of them the movements were very offensive; 3 other patients developed decided diarrhœa within the next week, and between June 14 and June 28 there were 5 cases of hemorrhage, 4 of which were fatal; 3 occurred in patients who had decided diarrhœa, 1 in a patient who was having two loose movements daily, and the other in one who had been having one loose spontaneous movement each day.

My service ended on July 31. In the meantime I saw nothing that I could consider to be evidence of gastro-intestinal outbreaks, beyond the fact that in my last week of service several patients developed diarrhœa of mild character, which had no other serious symptoms associated with it except in 1 case in which fatal hemorrhage occurred. Numerous patients had had some diarrhœa at the time they were admitted; but it was not especially offensive or troublesome in any, and in all of them it subsided in a few days under rigid dieting.

On April 1, 1906, I went on duty again, this time in the women's ward. During the next four months there were no perforations; and, except during a period that I shall particularly mention, there were only the following isolated cases of hemorrhage: April 4

(fatal), April 19 (mild), April 27 (slight), June 18 (fatal). Two of the above-mentioned hemorrhage cases had severe and persistent diarrhœa preceding the hemorrhage. Five other cases were admitted, in the course of the service, with a diarrhœa that was persistent in spite of careful diet and treatment. Numerous others had diarrhœa upon admission, which soon subsided. Except, however, for the one special period which I am about to mention, there were no instances of troublesome or complicated diarrhœa that developed after admission.

The exceptional period was from May 23 to May 30. In this time 6 patients that had severe diarrhœa had dangerous hemorrhages; and 5 of them died. Two of them were among the cases that I have mentioned as having had offensive diarrhœa upon admission. A third had had mild diarrhœa and slight hemorrhage before admission, and both diarrhœa and hemorrhage became more severe after admission. The other 3 developed the diarrhœa soon after the admission of those that already had this complication.

Besides the above-mentioned occurrences, after I had discussed some of these events with my colleague, Dr. Elliston Morris, he directed my attention on July 10, 1906, to the fact that he had in his wards at that time 2 cases with very bad diarrhœa, and 5 others with moderately severe diarrhœa, 2 of them having had persistent diarrhœa since admission. In 1 of these 2 it was exceedingly offensive. The other 5 had developed their intestinal disturbance in the ward, after the admission of the last-mentioned patient.

In all the instances that I have mentioned, the nurses that had the care of the patients that came in with diarrhœa also had to do with patients that subsequently developed diarrhœa. The milk was reasonably well excluded as the source of the trouble in all these instances, for during all of them it was either showing very satisfactory bacterial counts or was being pasteurized; and infection of the milk after its arrival in the wards was also pretty well excluded by the fact that the nurse who had charge of it and of its distribution had no other duties that gave opportunity for her to infect it.

In addition to these instances in which I have described the main course of events, I have, in looking over the records of the cases during 1903 and 1904, been able to determine that three of the little groups of hemorrhage and perforation that show in the chart that I shall subsequently present occurred in association with little outbreaks of diarrhœa that, the records state, developed in the wards. In two, at least, of these outbreaks of hemorrhage and perforation the accompanying diarrhœal outbreaks seem, from a study of the records, to have followed the admission of patients who already had severe diarrhœa; but this is so difficult to determine definitely from simply studying records, especially when their number is so very large, that I cannot be sure of the course of events.

The incidents that I actually observed sufficed, however, to make

me feel that most probably infections of the intestinal tract were being transmitted through the wards, and that the outbreaks of hemorrhage and perforation were largely consequent upon this. Considering that the food was an improbable source of infection, and that the mouth is the essential portal of entry, it seems to me highly probable that the infection had been introduced through the care of the patients' mouths; for they were bedfast, and unable to infect themselves through contact with other patients.

THE GROUPING OF HEMORRHAGES AND PERFORATIONS. That hemorrhages and perforations have a striking tendency to occur in groups or sequences has been repeatedly noted by writers. It has been especially remarked in relation to perforation. That this tendency is even more striking than is usually recognized, and that it appears to be due to circumstances existing in particular wards, since these accidents occur in groups in individual wards, seems to be pretty plainly indicated by the chart that I have prepared, which exhibits in their chronological sequence the hemorrhages and perforations that occurred in the men's and the women's wards during the years 1903 to 1906, inclusive, and in the first half of 1907. The total number of cases of typhoid fever during this time was 3677. The chart also shows that the great majority of both the complications under discussion occur in patients that have decided diarrhœa; that they are, therefore, closely associated with evident intestinal disturbance that is not an essential part of typhoid fever. In order that the chart might be simple in form, I have arbitrarily classed as having diarrhœa only those cases in which three or more stools were passed in the day. A considerable number of patients, however, had one or two very loose movements daily, and, therefore, really had diarrhœa; and if these cases were recorded as having diarrhœa, the relation of diarrhœa to hemorrhage and perforation would be still more striking. As the chart stands, however, the figures are: Hemorrhage without diarrhœa, 51; hemorrhage with diarrhœa, 136; perforation without diarrhœa, 15; perforation with diarrhœa, 45. It is apparent, likewise, that diarrhœa was much more prevalent in those that had hemorrhage or perforation in groups or sequences than in those that showed these accidents in isolated instances.

I have included in the chart as hemorrhages all cases in which blood was passed in noteworthy quantities. Mere traces of blood, whether occult or macroscopic, are not included. In order that judgment on this point and others might be uniform, I went over the histories of all the cases myself, in preparing this chart, except for about five hundred cases that Dr. Richard Hooker went over for me.

The next point to which I would direct attention in the chart is that there appear to be an enormous number of hemorrhages and perforations in 1905, and even more in 1906. This is, however, simply because the actual number of cases was extremely large.

In these two years we had 2389 cases of typhoid fever in the hospital, and there were 111 cases of hemorrhage. The percentage in which hemorrhage occurred was, therefore, only 4.6, which is only a little more than half of the frequency of this accident as it is given by most writers. The perforations in these two years numbered 36. They occurred, therefore, in 1.5 per cent. of the cases, which also is distinctly below their ordinary incidence. In the two preceding

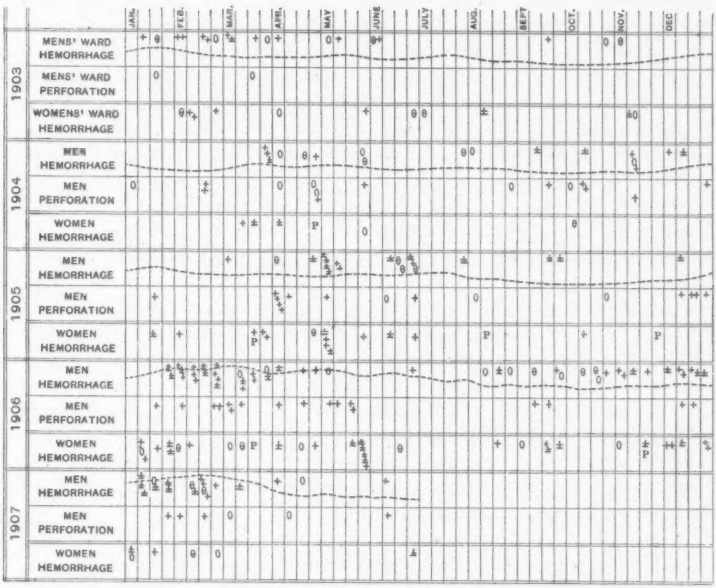

Chart showing the chronological sequence of hemorrhages and perforations in the typhoid fever cases in the men's and the women's wards of the Episcopal Hospital, Philadelphia, from January 1, 1903, to July 1, 1907. The months are divided into fourths (not weeks), in order that the divisions may correspond in the different years. The hemorrhages and perforations in the men are placed in contiguous but separate spaces; the perforations in the women were so few that they are simply indicated by the symbol P in the space for the hemorrhages. The symbol + indicates a non-fatal case with diarrhœa; ±, a fatal case with diarrhœa; 0, a non-fatal case without diarrhœa, and Θ, a fatal case without diarrhœa. Fatalities and recoveries in the cases with perforation are not separately indicated, as most of them were fatal.

years the percentages were: hemorrhages, 6.1; perforations, 2.1. These are still below the ordinary averages. I note this in order to show that there was no unusual frequency of these accidents, and that, consequently, there were no exceptionally bad conditions in this group of cases or in the circumstances in the wards which would make the things seen in this series of cases unusual and of no value as an explanation of ordinary conditions.

Since the hemorrhages were much more numerous than the

perforations, they give a much more striking picture. What I have to say refers chiefly, therefore, to the hemorrhages, although it applies almost as well to the perforations also; and, as I have said, the two, as they occurred in each ward, should, in this connection, be considered together.

It is at once noticeable that there are remarkable periods of more or less prolonged freedom from these accidents; then there are times when they occur wholly isolated; but the great majority of them occur near one another in point of time, either in prolonged sequences or in rapid succession in sharply demarcated groups. The groups do not show any tendency to occur coincidently in the two wards. In a few instances they overlap, and in one instance particularly, in May, 1905, striking groups were coincident in the two wards. In the latter outbreak, one might properly think that an especial severity of the group of cases that were being received was the occasion of so many accidents, or that the milk had given rise to the trouble; but the milk was in good condition at this time. Milk was, I think, undoubtedly the chief cause of the trouble, in the early part of 1903, in the men's ward; for it was at this time that the epidemic of milk infection that I have described occurred. The milk was probably, in part at least, responsible also for the succession of perforations seen in December, 1905; but, since the same milk was served in the two wards, it could not well be considered to have been the cause of the trouble in the other numerous groups that were not coincident in the two wards, nor can the trouble be well explained in such instances simply through a supposed difference in the character of the epidemic in the community.

The idea will at once suggest itself that the groupings were due simply to there having been exceptionally large numbers of cases of typhoid fever in the hospital at the time when exceptional numbers of accidents occurred. This, however, I think we may accept at once as being not true. To show this point I have introduced the broken line in the spaces for the hemorrhages in men. That running through the year 1906 and half of 1907 indicates the number of cases in the hospital in each week. In the other years I have introduced a similar line to indicate the number of cases admitted to the house in each month. In these last-mentioned years the weekly records of the number of such cases in the hospital were not preserved. The scale of elevation of this line is fifteen cases to each twelfth part of the vertical space. These lines indicate that there is no regular correspondence between the number of cases in the hospital and the number of abdominal complications. When the latter were very frequent there were naturally in some instances a very large number of typhoid cases in the hospital, but the number was, likewise, very large in some of the periods of prolonged freedom from accident, and the relation

between the number of cases and the number of complications is wholly variable.

Considering together, then, the facts that these accidents have a striking tendency to occur in groups or sequences, that they appear to occur in one ward at one time and in another ward at another time; that in most instances they occur in persons that have a disturbance of the bowels that is not an essential part of the disease; and, finally, that in a number of instances I was able to trace what appeared to be a clear relation between the admission into the ward of patients with offensive diarrhœa and the rapid subsequent development of diarrhea in a number of patients that had previously been free from it (these patients repeatedly showing either hemorrhage or perforation in groups), I believe that I had sufficient reason to suspect that secondary gastro-intestinal infections produced many, at least, of the outbreaks of diarrhœa and other complications, and that these secondary infections were passed around the ward.

Bearing of these Observations upon the General Conception of Typhoid Fever. This raises, of course, the still more radical question whether marked abdominal symptoms of any kind, including hemorrhage and perforation, are not ordinarily due to such secondary infections, more than to the typhoid infection itself. The severity of the abdominal symptoms in typhoid fever would then depend less upon the typhoid infection itself than upon the character of any secondary infections in the bowel. This, again, leads to a consideration of the question whether the ulcerative lesions in the intestine that are so nearly a regular feature of typhoid fever are not due to secondary infections more than to the typhoid infection itself, and whether the extent of the ulceration is not dependent chiefly upon the severity and character of any such secondary infections that may chance to gain entrance to the intestinal tract. I am, of course, not justified in going further than to point out the suggestiveness of the conditions that I have described in relation to these points. A definite conclusion in regard to such matters can be reached only by more exact studies, which must be chiefly bacteriological and subject to great difficulties. I may note, however, that these suggestions are, at least, in consonance with the fact that widespread infiltration of the lymphatic tissues occurs in typhoid fever, especially throughout the abdomen, but that destructive lesions in such tissues occur with very great rarity anywhere except in the intestines. There they occur almost always, and it is just there that varied kinds of bacteria are always present, so that the opportunity for some sort of secondary infection is always present. These suggestions are likewise in consonance with what we have learned regarding a number of other infectious diseases, in which the primary infection so often opens the way to a secondary infection that produces the most serious local lesions.

This question has more than an academic interest. Should the

above-mentioned suggestions prove correct, we must still expect that abdominal symptoms and accidents will inevitably occur in a certain number of cases, whatever we may do, for a variety of bacteria are always present in the intestines. But the practical bearing of the matter would be that, with strict precautions as to the food and general care of such patients, it would be possible to save many cases from unnecessary secondary infections, especially from those that are severe and dangerous; and it would thereby be possible to reduce the frequency of abdominal symptoms and complications. Indeed, whatever the relation of secondary infections may be to the ordinary lesions of the disease, I am, as I have stated, convinced through my experience with milk infections in typhoid cases, as well as through these observations which so strongly suggest ward-transmission of secondary gastro-intestinal infections, that the at least occasional occurrence of such infections must be reckoned with, and all reasonable precautions must be taken to prevent their occurrence through the food or the general care of the patients, particularly in hospitals, where there are large numbers of opportunities for the transference of such infections.

Pediatrists have learned through most painful experience, especially with infants, not only that milk is of enormous importance in producing infections of this character, but also that there is grave and constant danger of transmitting these infections from child to child. At present, there is no well-conducted infant's ward in which this danger is not combated by careful regulations. In spite of all care, transmissions occur occasionally; and in many such hospitals it is still customary to discharge infants as soon as they are in a condition that makes this at all safe, for fear that they may acquire such infections. The conditions in regard to adults present no essential difference, except that adult patients are ordinarily much less susceptible to such infections. Those adults, however, who are already more or less profoundly ill can hardly be considered to be vastly more resistant to such infections than are infants; and this is especially true of typhoid patients, whose digestive tracts are very easily disturbed. The conditions that I have described are closely analogous to those that occur in infants' wards. The fact that such conditions occur in infants' wards and the necessity for controlling them have, very properly, been accepted without definitely tracing their occurrence by bacteriological methods. The final evidence of their occurrence in these wards or in adult wards, and particularly the evidence as to the exact nature of these infections, must be given by bacteriological studies, but this evidence is not necessary to make precautions with adults seem reasonable, any more than it is with infants.

It will unquestionably be said by many clinicians that my experiences are at best unusual, and therefore not representative of ordinary conditions. This may be true; but it may, at the same

time, fairly be urged that I have especially searched for these conditions, and that I have had a most exceptional opportunity for observing a very large number of cases, and, therefore, for seeing things that would not be apparent in observing smaller groups, even though they occurred. The effect of this latter point is seen in the chart. The groups in 1905 and 1906 are very apparent and extremely striking. In 1905, in the men's ward, 21 accidents occurred in three definite groups; 5 others occurred in rapid sequence in December. Only 8 others, all told, occurred in isolated form during the entire remainder of the year, and 2 of these 8 occurred in close succession. The concentration of accidents in the early part of 1906 and 1907 is even more remarkable. In 1904, however, and especially in 1903, in which years the number of cases was much smaller, the groups are not striking. Nevertheless, looked at in the light of the conditions that were met with in 1905 and 1906, it is evident that there were groups, though smaller ones, in 1903, and especially in 1904. In the latter year, there are five little groups, in each of which 3 or 4 accidents occurred in about a week. The total of accidents in these groups was 20. In the whole remaining portion of the year only 12 accidents occurred, and several of these happened very close to the groups, and may have been really a part of the latter; 6 of them, indeed, occurred in pairs. Such occurrences may sometimes be simply coincidence. They may, however, quite as well be due to the conditions that I have suggested, and coincidence is a poor explanation for things that occur in a large proportion of instances, and is likewise an explanation that accomplishes nothing.

Transmission of Causes of Otitis Media. In addition to the matters that I have discussed, there are certain other types of ward infections, and precautions pertaining to them, to which I would refer. It has repeatedly been noticed in groups of sick children, particularly in those who have diphtheria or one of the acute exanthemata, that otitis media occurs with striking frequency, and this has sometimes been attributed, very properly, I think, to transmission of a special infection of the mouth-cavity among the cases at such times. The same thing occurs at times in adult patients that have various diseases, and, in addition to those diseases in which especial involvement of the nose and throat is a typical part of the disease, it is particularly seen in typhoid fever. In 1902 I had 7 cases of otitis media in typhoid cases in the course of two weeks, and in 1906 Dr. Stevens had among his cases a rapid succession of this complication. It is highly probable that when it occurs in a number of cases in the same ward within a short time, the infection causing it is transmitted from case to case, and it is most probably directly introduced into the mouth. The precautions that I have suggested regarding the treatment of the mouth would serve to reduce the frequency with which such infections also can be carried.

In addition to this, the nurses and doctors should, in treating ears that are already involved, adopt similar precautions with their hands, in order to avoid infecting the hands and thus transmitting the trouble. This applies to the treatment of cases of any kind in which otitis develops. It is, however, especially applicable to the wards of those hospitals which care for highly contagious diseases, such as scarlet fever, diphtheria, and measles, in which this complication is very common. In such hospitals, as in general hospitals, patients with the same disease are very often treated as if they were harmless to one another, while, when they have such complications, they almost certainly are not harmless.

Infections of the Skin. Infectious skin disorders have, likewise, given me some concern. I reported an experience that I had in 1903, in which a very peculiar vesiculopustular skin infection of considerable severity was evidently transmitted to five cases of typhoid fever in the same ward. It was due to a staphylococcus. On account of the situation in which it began in these cases, I suspected that it was carried by the bedpans or was inoculated by the nurses in rubbing the patients. I instituted precautions, which related especially to the disinfection of the exterior of the bedpans, the bath-tubs, and the gauze sponges used in giving sponge-baths; and I required the nurses to take particular care in sterilizing their hands after treating the persons of patients that had any sort of skin infections. This complication did not develop further in my ward, though the infection still seemed to be about the hospital, for it appeared later in another part of the house.

Previous to this I had twice seen a number of patients in succession develop, within a short period, a more or less extensive furunculosis. Since then, also, when I was not on duty, a man was admitted with a number of bullæ of the skin, which became pustular and then gangrenous. Two other patients, near by, developed the same condition within a few days. All three died. This condition, also, was evidently transmitted. More precautions than are usually taken are needed to prevent similar occurrences.

I have, since I saw the little epidemic in 1903, had the nurses that have charge of cases with any infectious skin lesions, even furunculosis, take especial care regarding the sterilization of their hands after treating them, and I prefer that, when looking after such cases, the nurses should wear rubber gloves that are kept for this purpose alone. This is intended to avoid carrying the infection to the skin of other patients (in rubbing them, etc.), and also to avoid the danger of transferring the bacteria from the skin lesions to the mouths of the same or other patients.

I have, likewise, had the gauze used for sponges boiled after bathing any patients, before it is used for others. That which is used for patients with skin lesions is kept for them alone, and the basins employed for sponging such patients are disinfected carefully

after their use. The question of the sponges would seem to be scarcely worth mentioning at the present day, but I know that it is often disregarded. Recently, in a prominent hospital in another city, an extremely old marine sponge was seen being used for a typhoid patient, and, upon inquiry, it was learned that it was used with many patients and it was not known that it had ever been subjected to any more drastic measures than ordinary washing. Marine sponges should not be used, for their supposed advantages are theoretical rather than real, and they cannot be successfully sterilized; and gauze sponges, from mere estheticism, as well as for safety, should be boiled after they are used. The boiling of the bedpans and other ward utensils is important also in relation to skin infections, as well as for the reasons that I have already mentioned.

If such cases as impetigo contagiosa appear in the ward, I isolate them and put a special nurse in charge; and I would do the same should any case such as those that I have described as occurring in little epidemics appear. Since I have taken precautions against such complications, I have had no multiplication of cases of extensive furunculosis and no evidence of the transmission of any other skin infection. These infections are rarely grave, though they occasionally are. They are always, however, distressing and, if severe, exhausting to the patient; they sometimes lead to bedsores, if they are in areas that are exposed to pressure; and there is always slight danger that serious general infection may occur from them.

Not less important than any of the conditions that I have mentioned is the proper care of such cases as pneumonia, tonsillitis, "influenzal" attacks, and, of course, tuberculosis. I think, too, that special attention should be given to dysentery and to any cases of gastro-enteritis or colitis that are apparently due to a sharp infection rather than to dietetic errors. So far as any of these require attention to their mouths (and pneumonia and tonsillitis cases always do), precautions similar to those that I have described should regularly be taken, in order to prevent carrying other infections into their mouths, and, likewise, to prevent transference from them to the mouths of other patients. The precautions against unnecessary soiling of the nurse's hands with excretions of all sorts, which I have described as applying especially to typhoid fever, are of importance in these cases also, and they should be regarded with particular care in dealing with all sputum, and with the stools of cases of gastro-intestinal infections. Drinking cups and other food utensils, thermometers, etc., that are employed with any of these cases should be for their individual use, or should be carefully sterilized after being used. Drinking vessels are best made of agate ware, in order that they may be boiled after they are used. The most important further points regarding the care of patients of the type now under consideration I may most readily mention under the general subject of the isolation or segregation of infectious cases.

Isolation and Segregation. Isolation of those infections which are received in general hospitals is, of course, an exceedingly difficult thing to carry out in a satisfactory manner, both because the present-day hospital plants are, as a rule, so constructed that they do not permit of it, and because it is very costly with any arrangement of the hospital, owing to the increased demands for nurses, the extra time consumed in other ways, and for other reasons. The problem that we have to meet is to make the best possible use of the plants that we now have, and to keep within reasonable bounds as to expense. Anything that we do that is at all effectual will cost somewhat more than methods that disregard this precaution, but physicians are quite as well justified in asserting the rights of their patients as are surgeons, and if they are convinced that their demands are just, they may properly insist with equal earnestness upon the insertion of their demands into the hospital budget. We can, however, with our present hospital plants, do much better than is now usually done, without going to very great expense.

In planning isolation and segregation measures, it appears to me that we should not endeavor to obtain simply the most ideal results possible with one or two particular diseases, but should attempt to cover the whole field of action in a manner that, while perhaps imperfect in relation to an individual disease, is such as to offer the greatest general security against all serious infections. I have, therefore, directed my efforts in a manner that differs somewhat from that which is customary at present among many of those who have urged isolation, especially as regards typhoid fever. The principle upon which I have acted is that at present we have little power, except through isolation or segregation, to prevent the transmission of those diseases which appear to be somewhat commonly transmitted through the atmosphere, or which we must at any rate, for safety's sake, still consider to be commonly transmitted in this way. We are in the same position with regard to those diseases which can be transmitted by very brief contact or by kinds of contact that are very difficult to avoid. On the other hand, with those diseases that are conveyed almost solely by means of intermediate persons or objects, or by direct handling of the patients' persons or excreta, we should, by adopting proper measures, be able to reduce the number of infections to so low a point that the danger from these diseases would be decidedly less than from the diseases that are or appear to be frequently air-borne. We cannot, at present, isolate them all; and a perfect system of isolation for all of them can scarcely be looked for in the near future. Indeed, any system of actual isolation of even all those that are of serious consequence will remain, for a long time, at least, a practical impossibility, and with some of them it will, I trust, be found to be unnecessary, for I believe that some of them can be prevented by less rigorous though more detailed measures. I am speaking, of course, of conditions in well-organized

hospitals, not of private practice. In the latter, because technical details cannot well be carried out, as a rule, and isolation often can, I believe in regular insistence upon as much isolation of all infections of consequence as one can secure.

Any plan adopted must depend upon the conditions in individual hospitals. That which I have gradually elaborated for my own period of service at the Episcopal Hospital depends upon the means available there. On the men's medical side, we have, on each of two floors, a large main ward, containing thirty-one beds; a small adjacent ward, containing seven or eight beds; and two smaller rooms, one of which will hold two or three beds, the other, four beds. These latter rooms were once used for private patients. They have recently been largely released by the managers for purposes of isolation. The quarters for the women are the same as those on one floor on the men's side, except that there is no small ward. The children occupy the upper floor on the women's side, and the arrangement on their floor is the same as on that for the women. Besides the quarters mentioned, we have heretofore used a series of unoccupied rooms on the upper floor of another building for the immediate isolation of any highly contagious cases that appear in the wards, and we now have a well-equipped isolating pavilion for such cases. On the men's side, the more acute cases are kept on the lower floor, while chronic and convalescent cases occupy the main ward on the upper floor. The side ward on the upper floor is kept for youths, who are best separated from the doubtful moral influence of the men in the ward.

Each day that I visit the wards I survey the situation of the cases in relation to one another, and determine that the arrangement of the cases is the best possible in my judgment, under the circumstances existing at that immediate time. The general plan followed is this:

When febrile cases of doubtful nature are admitted, they are put into the side ward on the men's side, into one of the side rooms in the women's quarters, until it becomes clear that they may safely go into the main ward or that they should go elsewhere. This reduces the danger from cases that would otherwise occasionally spread infections. When the wards are full, all the beds in this side ward are continuously in use, and suitable cases are moved into the main ward, while patients that are disturbing in the main ward (owing to delirium, outcries, incontinence, etc.) are, so far as possible, kept in the side ward, or, if very disturbing, they are put into one of the smaller rooms, if one is vacant.

Placing of Infectious Cases. Cases with recognized infections are placed as follows: Typhoid cases are put into the main ward, and cases of this disease are kept together, other patients not being put among them. Cases with acute gastro-enteric attacks, dysentery, and the like, are put into the side ward, if very ill. If not, they

are sent to the convalescent ward. The facilities for caring for those that are very ill are better on the lower floor; but I attempt to keep all such cases as well separated as possible from patients that have any diseases, especially typhoid fever, that render the transmission of such infections to them more probable and more dangerous. Nurses who have charge of such patients are warned to be especially careful about their hands.

Tuberculous patients are put into the four-bed room on the upper floor, this room being regularly reserved for them. It suffices for the number that we receive. It is almost undoubtedly possible to treat tuberculosis in a general ward with practically entire safety to other patients, provided that the tuberculous patients intelligently and conscientiously follow orders; but it is so uncertain whether they will do this or not, and they are so likely not to that I quite agree with the general view that they should be isolated. I think, too, that it is almost equally important on their own accou t to keep them out of the general wards, because "colds," attacks that fall under the group that clinically is usually loosely called influenza, and similar conditions, frequently turn up in the general wards, and tuberculous patients are very subject to these and are frequently seriously damaged by them.

Tonsillitis is rarely admitted. If it is for some special reason, or if it develops in the hospital, the patient is isolated in a separate room and a special nurse is put in charge of the case, both because it is often so difficult to exclude the possibility of diphtheria—and it is important to get this disease out of the ward, even before getting a definite report from the culture—and also because it is not just to expose other nearby patients to tonsillitis, which may be transmitted through brief exposure, and apparently through the atmosphere. If there is no room for actual isolation near the ward, the case, if at all seriously suspicious, is sent to the separate isolating building. If it is only slightly suspicious, the patient is treated in a corner of the L of the corridor, where he can be well separated from the others. If the ward is not very full, and the rooms are occupied, the case may be treated in a corner of the ward well separated from the other beds, a screen being placed around the bed of the tonsillitis patient to keep walking patients away from him. It is probable that the infection is transmitted atmospherically for only a short distance. Therefore, if other patients are kept at a considerable distance and strict care is observed regarding cleanliness of the hands, etc., it is probable that such patients may, with entire safety, be kept in the ward.

Danger from Pneumonia. Pneumonia has especially engaged my attention in regard to the question of isolation. My feeling is definitely that this disease needs to be isolated quite as much as any disease that is admitted to general hospitals, and, beyond all else, patients with pneumonia should be kept away from those who have

typhoid fever and other very depressing diseases. I have reported a little epidemic that occurred in my wards in 1903, the cause of which seemed to be clearly that a fatal case in the side ward that was very heavily infected had infected the surrounding area of the ward. The patient had been very delirious and had spat in all directions. The patients who became infected had been near him, but when the infection developed in them most of them had been moved to various parts of the main ward; consequently, it required some investigation to determine the source of the several cases of pneumonia that I found appearing in my ward in this instance. When determined, the side ward was formalized, and the trouble was entirely overcome.

It is perfectly well known that definite ward epidemics of pneumonia occasionally occur. Much of the testimony regarding ward and other epidemics I referred to some years ago[3] and need not repeat. Every clinician of experience has also repeatedly seen individual cases develop in hospital wards, especially in those who are near patients with pneumonia. I have, unfortunately, no definite records of the number of such isolated occurrences that I saw previous to 1903, but I can, from mere memory alone, recall three instances before that time in which typhoid patients developed lobar pneumonia when pneumonia cases were near them, and I can also remember a case of diabetes and one of recent mild apoplexy in which the same thing occurred when the patients were in beds next to pneumonia cases. All these five cases were fatal. Since the occurrence of the ward epidemic that I observed in 1903 I have been using the precautions in regard to isolation or segregation that I shall describe, and—whether as a consequence of this or not, I cannot state—I have seen but two other cases of lobar pneumonia develop in patients in my wards, and one of these occurred after a patient with unrecognized pneumonia had, in my absence, been placed in a bed between two typhoid patients. I have, therefore, no important recent observations of my own that are pertinent to add to these earlier cases. I think, however, some evidence of occasional transmission of pneumonia could be found in any large hospital if actually looked for. In examining the records of the typhoid cases I found casually that in the last week of April, 1904, when I was not on duty in the men's ward, there occurred four fatal cases of lobar pneumonia in typhoid patients within a week, in a limited area of this ward, after pneumonia cases had been placed near or among them. One of my colleagues has also informed me that since that time he has had six ward cases of lobar pneumonia within two weeks, in the same circumstances. I have knowledge, also, of similar occurrences within the last four years in several other hospitals in Philadelphia. In one of these, the pneumonia infections were

[3] Therapeutic Gazette, 1904

so numerous and persistent that the ward was closed and formalized. In another (the Philadelphia Hospital), there were so many infections as to lead Dr. Martin and Dr. Biggs to set aside a ward for pneumonia cases. Most of the infections mentioned were in typhoid patients, who are extremely non-resistant to this infection.

Such infections not infrequently occur in individual cases, and I think that there can be little doubt that ward infections with pneumonia are, all told, as frequent as are ward infections with typhoid fever. They are certainly much more dangerous, for a large proportion of them are fatal, especially when they occur in patients already seriously ill—as, for instance, in typhoid patients. I feel, therefore, that pneumonia, first of all the acute diseases received in general hospitals, should be isolated in some degree. The literature on ward epidemics shows that most of the cases have developed in patients who were near those who had pneumonia. If the patient is tractable, therefore, so that the sputum can be properly cared for, it is very probable that the danger becomes extremely slight if he is kept at a distance from other patients, even though he be still in the same ward, provided that precautions are also taken against transferring the organisms to others by means of the hands of the attendants, the drinking vessels, etc., and that extreme cleanliness is used in regard to the ward floor, etc. Safety in cleansing the ward will probably be greatly furthered through the introduction of the vacuum system of cleaning.

My custom, then, is to place pneumonia cases in one of the rooms near my wards, so long as I have space for all of them there. If I have at one time too many cases of pneumonia, and of other kinds that need isolating, to permit of this with all the pneumonias, I put the mildest and most tractable of the latter in the ward, separating them as far as possible from other patients, especially from those with typhoid fever or other diseases that greatly reduce resistance, and, if there are any vacant beds, placing these between the pneumonia patients and the nearest other patient. I also screen the beds of the pneumonia patients because this probably helps to prevent the spread of infection, and it keeps other patients away from them. It also has a good moral effect upon the ward staff, as a reminder of the need of care. Delirious or intractable pneumonia cases are always kept out of the wards.

Isolation or Segregation of Various other Common Infections. I segregate in the ward, or, if I have space, isolate, cases that develop so-called "influenzal" attacks, whatever bacteria appear to be causative. When diagnosticated as such, they are not ordinarily admitted when such conditions are epidemic, if we can humanely avoid it, because it would be impossible to isolate large numbers. If they must be admitted, they are isolated, if possible, or, at any rate, segregated. So far as possible, I also separate from others in the ward all patients with ordinary "colds." Whenever new cases

with any infectious disease are admitted to the ward, I attempt to see myself that the position in which they are placed in the ward or in isolation is the most satisfactory possible in the circumstances existing at that time. I have already noted what I do with skin infections.

I believe that such a system is, under our present working conditions, likely to accomplish more good than simply directing our attention to one or two important diseases and permitting the rest to be mixed up together. I have not followed the recent trend, and attempted to isolate typhoid fever, because I believe that, with the precautions in the care of this disease that I have described, it is probably possible to reduce the danger of its transmission to so low a point that it is very slight, and less than that from those other diseases which I do isolate. I prefer to use my isolation space, first of all, for other diseases, particularly pneumonia. I have also had so many typhoid cases that I could not have isolated them unless the main ward, and often even more space, had been used for them alone, and this would at frequent intervals have left a large part of our space unoccupied, and would thereby have worked hardship to a large number of patients seriously in need of hospital care, who would have been excluded.

In the last half of 1907, the introduction of a supply of filtered water has marvellously reduced the number of our typhoid fever cases. Instead of from 50 to 150, there are now usually from 10 to 20 typhoid cases in the hospital at one time. I may find in future services that I can isolate these cases, and, if so, I shall hasten to do this, but I shall still give preference in isolating to diseases that are apparently capable of being conveyed through the atmosphere. The danger that children may acquire typhoid fever in a hospital ward is, however, so very considerable, and contact infection is so very hard to prevent with any children that are on their feet, that the measures I have suggested as suitable for adults would be much less effectual with them, and with them, I think, isolation should be rigidly insisted upon.

One important way in which adult patients acquire typhoid fever is, undoubtedly, through helping about the ward. While preparing this article I have seen a walking patient carry a bedpan from a typhoid case and immediately afterward start to eat his dinner without having washed his hands. This is an example of common occurrences. Orderlies, who are employed for such purposes, must take their chances, after having been duly warned, but it is not proper to permit patients to endanger themselves in such ways.

Apparent Effects of Regulations. My faith in the system of asepsis and partial or complete isolation that I have described is at least not lessened by my experience in recent years, and particularly by my service in 1907 at the Episcopal Hospital, since this paper was prepared. During this latter service I had this system, for

the first time, in fairly complete order. While there were, of course, many little ways in which it was imperfectly carried out, I escaped, for the first time, almost all evidence of avoidable ward transmission.

As I have already stated, I had seen no evidence of the transmission of pneumonia infections since I instituted precautions against it, whereas during the several years before that time I had seen evidence of it each year, and in one service I had had a little epidemic. In 1907 I had a large number of cases of pneumonia for some time, but there was no evidence of ward infection with this disease, except in one case. This was the instance mentioned in which a patient with an unrecognized pneumonia had, in my absence, been put for two days among the typhoid cases. Within these same years in the same wards two of my colleagues, at a time when they were placing pneumonia cases among or in proximity to their typhoid cases, had a rapid series of cases of lobar pneumonia in their typhoid cases, and there were, in addition, a considerable number of individual cases.

As to typhoid fever, I had had, in each of the four preceding years, at least one definitely recognized case of ward infection with this disease, in spite of sufficient protection of the food. In 1907 I had no ward infection with typhoid fever. This, however, may have been partly due to the fact that I had only about two-thirds of the number of typhoid cases that I had seen, on an average, in each of the preceding four years.

For three years before, after instituting precautions against them, I had seen no evidence of the transmission of skin infections, and I saw none in 1907. In these four services there had been in the wards a number of isolated cases of virulent furunculosis and two isolated cases of the peculiar vesiculopustular infection that I had previously seen spread rapidly. None of those seen in the past four years spread at all.

In each preceding year I had seen little outbreaks of diarrhœa and sudden groupings of hemorrhage and perforation in typhoid cases; the diarrhœa developed in a considerable proportion of instances in those that had been free from it on admission, and a large proportion of the hemorrhages and perforations occurred in these latter cases. In 1907 I had an exceptional number of typhoid cases admitted with severe and persistent diarrhœa, and I saw no evidence of the transmission of this, and had no groupings of abdominal complications. Indeed, I had only one severe case and two very mild cases of hemorrhage and two perforations, one of the latter occurring unexpectedly in a convalescent. I saw during this time 116 cases of typhoid fever.

This satisfactory state of affairs may have been partly due to chance. I think, however, that it may fairly be considered to have been due in part to the system used.

PROTECTION AGAINST FLIES. There are several other points to which I would refer in closing. The danger of the transmission of diseases by insects is now so well known that screens in ward windows and doors are becoming, in well-arranged hospitals, a pretty thoroughly recognized necessity, in order to protect uninfected patients as well as the surrounding community. Even, however, in those wards that are screened one can frequently see many flies, and they, of course, get well covered with infectious material. They should be destroyed as completely and as rapidly as possible.

DIVISION OF WORK AMONG THE NURSES. Another point that is occasionally of much consequence and that needs supervision is the division of work among the nurses in the ward. I have found a number of instances in which the plan used was very bad. I have mentioned the worst instance, in which the nurse's duties included the preparation of the liquid diets and the disinfection of the soiled bedclothes and the stools. Another arrangement that, because it was less obviously dangerous, is a better illustration of the need of critical supervision of this matter was made apparent through a series of little ward epidemics of diphtheria in the main men's medical ward at the Episcopal Hospital. An investigation of this matter by the staff finally developed the fact that, owing to pressure of work, it had for some time been arranged that a nurse occupied in the out-patient department half the day should spend the other half in helping in the men's medical ward, and this nurse, it was found, was the one who had worked in the nose-and-throat dispensary. In the last outbreak of diphtheria she had evidently carried the infection, for it was learned that she had had a sore throat for a day or two before diphtheria appeared in the ward, and investigation showed her trouble to be an atypical diphtheria. Not improbably, the previous outbreaks had been due to the same cause. Of course, no scheme can be suggested for the nurses' duties that would be suitable in all hospitals. The arrangement in each ward should, however, be known by the visiting physician, and its suitability judged of by him.

MORE EMPHATIC WARNING OF NURSES. Finally, I would urge that greater attention be given to instructing the nurses regarding the very serious danger that they are in from typhoid fever, and that more detailed and constant attention be given to ensuring their use of proper precautions, for they are often exceedingly careless as regards themselves. We have had an unfortunately large number of cases of typhoid fever in nurses at the Episcopal Hospital, and I urged the adoption of some of the measures that I have described quite as much on their account as because of the patients. I think, however, that the figures I have given show that, as compared with the great number of typhoid cases that they have had to care for, and as compared with the figures elsewhere, the number of nurses that we have had sicken with this disease is really gratifyingly small.

This must, I think, be attributed to the fact that Miss Payne, who has been chief nurse during this period, has had a serious and constant sense of the importance of the matter, and has devoted much attention to it. It is, likewise, due to her responsive interest in such matters and to her intelligent direction that I have been able to put into operation the plans that I have had in mind, and I am indebted to her for many suggestions, as I am likewise to Miss Marion Smith, who has adopted many of the same regulations at the University Hospital.

The scheme that I have described may seem somewhat formidable, when viewed as a whole. The regulations do add to the work of the wards, and this cannot be avoided. It is difficult to have them used in satisfactory detail, and I have not yet been able to secure or to demand their use in entirely satisfactory form. This is, however, to be expected, especially at first. They have, in some part, been used for several years past during my services at the Episcopal Hospital, and in chief part during my last service. In considerable part, they have been used for some months past at the University Hospital. The burden that they have caused in these institutions has not been heavy.

I am fully convinced that neither these nor any other systematic regulations will be of much profit to anyone unless the visiting physician takes constant care to determine that the details are carried out continuously and as accurately as possible, in the manner that is best suited to the wards in which he does his work.

THE NATURE AND THE MANAGEMENT OF DIABETES MELLITUS.

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Diabetes mellitus was known to Celsus and Aretaeus in the first century A. D., and was described and named by Galen about 200 A. D.; but though so long recognized its etiology is not even yet perfectly understood. Though often accompanied or perhaps caused by pathological conditions, diabetes is really a disturbance of metabolism, rather than an actual disease as we understand that term.

Diabetes mellitus and glycosuria are not interchangeable terms. The name diabetes mellitus should be restricted to such cases as have sugar in the urine which cannot be removed by any dietetic or medicinal treatment. On the other hand, glycosuria, while a symptom of diabetes mellitus, may be only an indication of functional disturbance, alimentary, nervous, etc.

If this symptom of sugar in the urine is removed by regulation of the diet, hygienic or climatic changes, or by medicinal treatment, the condition is that of glycosuria and not diabetes mellitus, although the fact that sugar readily appears in the urine from indiscretions in diet, or perhaps from normal diet, shows that there is a disturbance of metabolism which we may call, for want of a better term, insufficiency of the pancreas, or, perhaps better, of the organs engaged in sugar metabolism. Such insufficiency in sugar metabolism, or clinically recurrent glycosuria, should be considered a danger signal, as the condition is often a forerunner of real diabetes mellitus.

The principal source of glucose is the carbohydrates of the food. They are changed during digestion into maltose and later absorbed as dextrose; the dextrose goes in the blood to the liver, where it is stored. During the process of digestion the portal vein contains more dextrose than any other blood of the body, thus proving that the liver retains what reaches it and gives it out as glycogen as it is needed.

The muscles of the body, on a mixed diet, contain about 1 per cent. of glycogen, though the muscles of the embryo contain much more, while the liver of the embryo contains but little. The object of this glycogen seems to be to produce energy as exemplified by heat and work; about three-fourths of the heat of the body is produced by the skeletal muscles. It has been proved that glycogen can be made from proteids, and, clinically, we know that in cases of diabetes mellitus the entire withdrawal of sugar and other carbohydrates from the food diminishes but does not abolish the glycosuria. If the carbohydrates are removed from the blood of an animal and the animal energetically exercised, glycogen will disappear from the liver, showing that the reserve glycogen is used for muscle activity. On the other hand, if muscular activity is diminished by narcotic drugs, as chloral, glycogen is again found in the liver, showing positively that it can be made from proteids, but ordinarily not in larger amount than is needed by the muscles. The administration of fat does not increase the amount of glycogen in the liver, but the administration of glycerin retards its elimination and leads to its being stored in the liver.

Some drugs under certain conditions can produce temporary diabetes, such as phosphoric, lactic, and hydrochloric acids, as well as strychnine, phosphorus, arsenic, and carbonic oxide. Just exactly how these drugs cause this glycosuria is not known.

Increased secretion of the thyroid gland may cause sugar to appear in the urine. This is often noticed in Graves' disease, and can often be caused by the administration of thyroid extract.

Disturbances of the central nervous system, especially of the floor of the fourth ventricle or the medulla oblongata, may cause such metabolic disturbance that glycogen in the liver is formed into glucose rapidly and appears in the blood in such quantities that all cannot

be used, and hence it is rapidly excreted in the urine, as the blood can tolerate only a small amount of glucose.

It is quite possible that the afore-mentioned acids disturb the liver function, hence the glycosuria; while the strychnine and phosphorus may disturb the nervous system sufficiently to be the cause of the glycosuria; and arsenic is certainly a stimulant to the thyroid gland.

Complete removal of the pancreas is followed by severe diabetes mellitus as well as by the appearance of acetone, diacetic acid, and β-oxybutyric acid in the urine, the latter forerunners of acidemia and coma. If a portion of the pancreas is left *in situ*, or a small piece of the pancreas is transplanted beneath the skin or into the peritoneal cavity, and the rest of the pancreas removed, diabetes does not occur, while if this transplanted portion is later removed, diabetes will occur. Such experimentation positively demonstrates that the pancreas has an internal secretion which plays an important part in sugar metabolism. Unfortunately the feeding of pancreas to animals from which the pancreas has been removed, does not stop the glycosuria.

Experimentation by Herter, of New York, and Croftan, of Chicago, has shown that disturbances of the suprarenal glands may allow sugar to appear in the urine, that is, that massage of the suprarenals or hypodermic or peritoneal injections of suprarenal extract, if in sufficient quatity, can cause glycosuria, even on a starch-free and sugar-free diet. This seems to mean that too much oxidizing stuff, which the suprarenals secrete, is forced upon the pancreatic and liver metabolisms at once and glucose in too large an amount circulates in the blood, the result being its rapid excertion by the kidneys.

Pathologically a large number of cases of diabetes mellitus show disease of the pancreas, and especially the part of the glandular portion of the pancreas which is ductless, the islands of Langerhans. These islands of Langerhans are the part of the pancreas that furnishes an internal secretion or a ductless secretion, a secretion which reaches the blood by absorption. It is quite different from the part of the gland that furnishes the pancreatic digestive juice that reaches the intestines through the pancreatic duct.

Pathologically we cannot always account for the glycosuria that is present in patients who have no apparent histological disease of the pancreas. We must explain these cases on the hypothesis that there is a disturbance in the relationship between a part of the secretion of the suprarenals, the secretion of the islands of Langerhans of the pancreas, and the glycogenic function of the liver cells. If such disturbance of these secretions is permanent or unchangeable, diabetes mellitus develops. On the other hand, if the relationship of these secretions is such that a diminished amount of carbohydrate or sugar can be properly metabolized without too much glucose occurring in

the blood and, therefore, appearing in the urine, there is a condition of sugar metabolic insufficiency only. Doubtless, like many other organic insufficiencies, after a period of rest the gland or glands or parts of glands may recuperate or new glandular tissue develop to do sufficient work, and the patient is again able to metabolize the normal amount of starches and sugars.

It should not be lost sight of that reflex nervous disturbances, even if the disturbances are functional, can interfere with these secretions so that sugar may appear in the urine. The disturbance being removed, the sugar will disappear and the recovery may be permanent. Acetone in the urine in any amount is pathological. In starvation and in rigid proteid diet acetone may occur with perhaps diacetic and later β-oxybutyric acids. The free administration of carbodydrates will cause the acetone entirely to disappear, while large amounts of fat and butter increases it, but there is no relation between the sugar excretion and acetonuria in diabetes.

It has been shown that the glycosuria sometimes following ether or chloroform anesthesia and alcoholic intoxication is not due to these drugs, but to some metabolic disturbance caused by them, probably to the dyspnœa, that is, possibly the carbon dioxide poisoning.

It seems to be a fact that the greater the amount of proteid diet, especially with the absence of carbohydrates, the more acetone appears in the urine; the greater the amount of fat ingested the more acetone appears in the urine; while the greater the amount of carbohydrates ingested the less acetone appears in the urine.

The diabetic seems to be unable to derive the proper amount of energy from his carbohydrate food, and, consequently, feels the necessity of eating more proteid and fat, which, if his system is able to digest, keeps up his nutrition. If his digestive or excretory organs become deranged his nutrition begins to fail, as he cannot take enough proteid and fat to satisfy the energy needed and ordinarily produced by carbohydrates, and he then begins to emaciate. It is thus understood why the diabetic eats so much, and why, sooner or later, he emaciates. The enormous excretion of urine seems to be the only cause of the enormous ingestion of water.

The other symptoms of diabetes, such as the tendency to a dry skin and, hence, to eczemas, can be due to the enormous loss of water from the tissues. The predisposition of diabetics to infections is probably due to the fact that the blood, with its increased amount of sugar, is unable normally to produce its opsonins and antitoxins.

Diabetic coma is not due to acetone or glucose, but is due to the diminished alkalinity and finally acid condition of the blood, an acidemia due principally to diacetic and β-oxybutyric acids. The warnings that such a condition is about to occur are headaches, general nervous irritability, and the presence in the urine of one of these acids; quite likely at the same time the amount of sugar and of

the nitrogen may be actually diminished. The urine may show at this time a large amount of ammonia, which is probably formed by the increased acids in the urine breaking up the alkalies.

An absolute withdrawal of carbohydrates from the food of patients having true diabetes mellitus will always increase the acetone and diacetic acid and often the ammonia and β-oxybutyric acid, and toxic acidemia and coma become imminent. Hence it is unjustifiable, sugar having been discovered in the urine, to withdraw the starches absolutely or too rapidly from the diet. The condition may be carefully and safely studied by the gradual diminution of starches and sugars and the frequent examination of the urine to see if it is free from diacetic acid, which not appearing, and the carbohydrates being reduced to a minimum and sugar disappearing from the urine, show that the patient is suffering from functional or dietetic glycosuria and not from diabetes mellitus. On the other hand, diacetic acid appearing, the carbohydrates should be increased until it disappears. Also, without the appearance of diacetic acid, if sugar persists in the urine after several days of rigid carbohydrate-free diet, the sugar being formed from the proteids ingested, renders such rigid diet unjustifiable, as the enormous amount of proteids and fats that must be consumed to produce heat and energy so overwork the proteid metabolism of the body as to cause sooner or later serious indigestions, organic insufficiency, and finally organic disease, to say nothing of the danger of sudden acidemia and consequent coma.

While the ammonia in the urine is perhaps the most accurate indication of the diminished alkalinity or the acidemia present, its estimation is chemically much more difficult than the simple test for diacetic acid, which latter will positively indicate the danger of coma occurring.

If a few drops of a 5 per cent. solution of ferric ammonium sulphate is added to the suspected urine, it will cause a crimson color to develop if diacetic acid is present. The more intense and deeper the color, the more of the acid. Diacetic acid being discovered, carbohydrates should be given immediately and in considerable amount, and the coma will generally, at least temporarily, be averted.

Diabetes seems to be on the increase, possibly from our increased nervous tension giving more hours daily and more days a week of nervous tensity, which sooner or later may cause nervous irritability. The Hebrew race seems to be the most predisposed, while the colored race is rarely attacked by the disease, again possibly an indication of the nervous element, there being no race showing more nervous energy than the Jewish, and no race such phlegmatic laziness as the colored. It is a rare disease in infancy, and the tendency and frequency gradually increase until the most deaths occur between fifty and sixty years of age, again the period of exhaustion from continued nervous energy. The disease also occurs more frequently

in men than in women. Heredity is an important factor, and is quite frequently an apparent cause in children, while other families show a tendency for it to develop with obesity later in life. Perhaps 20 per cent. of the cases show a family tendency or inheritance.

While it is stated that diabetes mellitus can be an acute disease, I feel that it had better be stated that glycosuria can be an acute serious condition caused by some infection or serious injury, while true diabetes mellitus seems almost invariably to be a chronic disease.

The most frequent complications are those of the skin, as pruritus, eczema, erythema, boils, and carbuncles, while gangrene readily follows slight injuries to the extremities. Ulcerations in the mouth and caries of the teeth occur. There is often loss of the patellar reflexes, and much muscular debility. There may be paralysis of the ocular muscles, and cataract and diabetic retinitis are not infrequent occurrences. Tuberculosis and all infections are readily acquired by these patients.

Without serious complications the disease runs a rapid, fatal course in children, while adults, especially if obese, in which condition the prognosis is the best, may live for years. Death is usually caused by diabetic coma, which is generally preceded by ever increasing indigestions and progressive emaciation.

Treatment. The general management of the case is essential; medicinal treatments are of very secondary importance. The diet should be regulated best to suit the individual case, and this can only be discovered by gradual changes in the diet and frequent examinations of specimens of the twenty-four hours' urine, together with a careful consideration of the efficiency of the digestion and the maintenance of the body weight. Whether or not the patient should remain in the climate in which he is, is a question that can only be settled individually. The proper diet, a modification of mental worries, a modification of work and exercise, with good air, proper warm bathing to keep the skin healthy, perhaps massage, and the discovery that the case is one of glycosuria rather than actual diabetes, may allow a patient to remain at or close to his ordinary life. If, on the other hand, the case is one of diabetes mellitus and there is the money ability or opportunity to move to a warm climate which is not subjected to great changes of temperature, the patient should be advised to take advantage of such ability. Experience has shown that sudden changes from warm to cold and excessive muscular exercise, as is also true in chronic nephritis, may precipitate a diabetic coma in the one instance and a uremic coma in the other. Hence, we should cause our patients to defend themselves as much as possible from these dangers.

The actual diet or the gradual modification of the diet is too well understood to require more than brief mention. As we diminish the carbohydrates and heat-producing substances, we must add fats in the form of butter, cod-liver oil, or pure olive oil, if it can be

secured. I have not a doubt that many a patient with diabetes mellitus has been hurried to his grave by rigid starch-free diets. I also believe that the fraudulent fact that most so-called starch-free gluten foods contain starch has allowed many a diabetic to live months longer than a starch-free diet would have allowed. My own method of diet is to remove all starches and sugars except bread for a time; to allow at first plenty of bread, and then gradually to reduce it, depending upon the amount of sugar in the urine and whether or not there is diacetic acid. If diacetic acid does not occur and the sugar continues to diminish, I reduce the bread to a minimum. If I find that a certain percentage of sugar continues in spite of the diet, I find the amount of starch that is needed to keep the patient from hunger and prevent him from eating enormous amounts of proteid and to keep up his weight. I then may allow him a slice of bread at each meal, or he may substitute for the bread a potato, allowing him to eat only a certain small amount of starch at each meal. If he complains of feeling cold, as patients on such a diet often do, I may even allow him an occasional lump of cane sugar, acting on the theory that if his metabolism continues to produce glucose from proteid, he had better have some starch or sugar to produce it easily and perhaps satisfy the mal-metabolism with less disadvantage to the system. As above stated, if diacetic acid is found in the urine, the starches should be increased immediately until it disappears. Potatoes are often more perfectly metabolized by glycosurics than are breads.

As to the feeding of organic extracts, the only ones to consider would be pancreas and suprarenal. I have never found any results from feeding pancreas. I have sometimes found good results from feeding suprarenal tablets, two, three, or four three-grain tablets a day. I have never been able to eliminate the sugar from the urine in a true case of diabetes mellitus by giving the recommended digestive ferments, although it is quite probable that some of the best of these ferments will allow a patient with alimentary glycosuria to digest and tolerate more carbohydrates than could otherwise be used without glycosuria occurring.

Various drugs have been found to modify the sugar excretion in diabetes, such as salicylic acid, salol, antipyrine, and various benzoate and guaiacolate combinations, but as no one of these drugs can be taken for any length of time without doing harm to the system, any temporary decrease in the amount of sugar is a matter of unimportance. Certain it is that prolonged use of these drugs will do harm.

I cannot believe that arsenic, which has been recommended, is often if ever justifiable. Personally, I have found it to do more harm than good. Opium, or one of its alkaloids, as morphine or codeine, often reduces the excretion of sugar, but it is also a sure method of causing the opium habit, and as soon as we understand that it is not

the excretion of sugar in the urine that is doing the patient harm, we will not use such drugs to diminish that excretion, that not being the disease, but only an indication of it. The action of morphine when taken for a long time has been shown to be probably upon the suprarenal glands. Profound poisoning by morphine seems to inhibit the activity of these glands, hence, repeated doses probably act mildly and continuously upon them. While this may reduce the formation of glucose, it can probably rarely, if ever, modify the course of the diabetes, and certainly adds many uncomfortable symptoms, such as increased dryness of the skin, loss of appetite, constipation, and all the nervous discomforts of the morphine habit.

It diabetic coma is imminent, besides pushing the carbohydrates, alkalies should be given, and there is probably none better than the simple bicarbonate of sodium. The danger being passed, simple alkaline mineral water may be administered for some time, especially if the bicarbonate of sodium causes indigestion. If coma has developed, venesection and the withdrawal of considerable blood, and then the administration intravenously of bicarbonate of sodium solutions, has caused the awakening of the patient; but there seems to be no permanent cure, the patient again relapsing into coma, in from twenty-four hours to two or three days. Physiological saline solution has been found as valuable as the bicarbonate of sodium solution for this purpose, while the alkali may be administered by the stomach or rectum.

THE IMPORTANCE OF THE EARLY DETECTION OF ANEURYSM OF THE AORTA.

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The attitude of the general practitioner today toward thoracic aneurysm may be compared to his attitude a few years ago toward the recognition of pulmonary tuberculosis—he was satisfied to recognize the disease when it was fully developed. It was not altogether his fault, but rather that of the text-books, which, in giving a single picture of the whole disease, naturally described the typical or developed cases. These cases must be regarded, in tuberculosis or aneurysm, as in the advanced stages, and as very hopeless from the point of view of treatment.

We have learned our lesson in tuberculosis. We now know the importance of recognizing it in its incipient stage, when we can greatly benefit, and often cure, our patients. This matter has been

presented to the profession thoroughly, and the picture of tuberculosis that the general practitioner carries in mind today is of the incipient or early stage of the disease—not the advanced stage. He is eager to recognize this early stage, and, as a result, the numbers of "arrested" or "cured" cases are multiplying rapidly.

Aneurysm of the aorta is not as common a disease as tuberculosis, but it is even more hopeless, and it causes great suffering. It is the more hopeless in proportion as it is advanced. Consequently, if we are to benefit our patients, we must discover it early—the earlier better. It may be noted in passing that aneurysm of the arch of the aorta is much more common than is generally supposed.

I have drawn a comparison with tuberculosis not because it is as important a disease, nor because the two diseases are similar, but because the general practitioner has been taught to do today for tuberculosis just what I hope to see him do in the future for aneurysm. He is keeping tuberculosis in mind; he is improving his technique; he makes careful examinations of the chest with the disease in mind; and he realizes the importance of its early recognition. It requires no more training or skill to recognize aneurysm early than it does in the case of tuberculosis. Therefore there is no reason why it should not be detected early, if the general practtioner will only keep it in mind and will examine with the same thoroughness as he does for tuberculosis.

Some forms of aneurysm cannot be recognized in their early stages. This is especially true of those situated at the very beginning of the aorta, at or just above the sinuses of Valsalva. When small, they give no symptoms and cannot be distinguished by physical examination. By the time they get large enough to be so distinguished they usually rupture into the pericardium, and sudden death is the first evidence of their presence. Aneurysms of the descending arch and the descending thoracic aorta can rarely be recognized in an early stage. This is due partly to their location deep within the chest, where they are inaccessible to physical examination, and partly to the fact that no important structures are pressed upon until the aneurysm has attained a considerable size, and until this point is reached there are no symptoms. Therefore, they are advanced when recognized clinically.

From a clinical standpoint, then, we are concerned essentially with aneurysms of the ascending arch and of the transverse arch. These are accessible to physical examination, and they can be detected much earlier than is generally supposed. Moreover, this part of the aorta is the commonest site of aneurysm.

As in the case of most diseases, the recognition of the condition will be based partly on symptoms and partly on physical signs. There is a difference between aneurysms of the ascending arch and those of the transverse arch in this respect, as pointed out by Broadbent. In the case of the ascending arch the aneurysm is near the surface,

and easily accessible to physical examination. It is, moreover, surrounded by structures that can easily be pushed to one side without serious results. Consequently, symptoms from pressure rarely arise until the aneurysm has attained considerable size. In the earlier stages, therefore, we detect these cases by physical signs rather than by symptoms, and Broadbent calls these aneurysms of the ascending arch the "aneurysms of physical signs." In the same way he speaks of the aneurysms of the transverse arch as "aneurysms of symptoms." These latter are more deeply situated and are less easily detected by physical examination. Before they get large enough to be easily detected by physical signs they press upon some of the important structures by which the transverse arch is surrounded and thus give rise to early symptoms.

Percussion furnishes the most important of the physical signs of aneurysm of the arch of the aorta in the early stages. When inspection and palpation are of use, the aneurysm must have reached the chest wall—and is already in an advanced stage. I wish to emphasize this point, that it should be our aim to detect aneurysm of the aorta *before* it is visible or palpable—even by the most careful examination. Auscultation is of little value as commonly employed. The physician is too apt to base his decision on the presence or absence of a systolic murmur over the site of the suspected aneurysm. A murmur is frequently absent, even when the aneurysm is large, and one of the few dogmatic statements that can be made about aortic aneurysm is—*never* exclude aneurysm because a murmur is not heard.

The importance of percussion in determining an enlargement of the arch of the aorta is shown by the fact that by practice we can determine the outlines of the right side of the arch fully as accurately as we do the outline of the left border of the heart. We can do this in a normal individual, and it is naturally easier when an aneurysm is present, which brings the vessel wall nearer to the surface. For this purpose percussion must be extremely light; otherwise the bony structures of this region are thrown widely into vibration, and our note is that of the sternum and ribs backed by the resonating power of the air-filled lung tissue. Very light percussion is all that is necessary, because the aorta lies near the anterior chest wall. Percussion in the interspace is preferable to that over the rib. Before accepting aneurysm as the cause of a change in the percussion outline, we must remember that deformity of the chest or spine may cause displacement of the aorta to the right, or retraction of the lung tissue may leave it uncovered to an abnormal extent.

Under normal conditions the transverse dulness at the level of the manubrium extends 2 or 3 cm. to the right of the median line of the sternum, and 1.5 to 2.5 cm. to the left of the median line. The total transverse dulness is 4 to 5 cm. We may consider anything above 5 cm. as more or less abnormal and requiring consideration. On the right we can make out a curved line of dulness in the adult,

as characteristic in outline as either border of the heart. If this border of dulness is farther to the right than normal, and if the normal curve is increased, we know there is an abnormal condition of the aorta—probably an enlargement or aneurysm.

The aorta is nearest the anterior chest wall at the junction of the second right interspace with the sternum. From this point, as it arches over to the left, it also plunges deeper into the thoracic cavity, and soon gets beyond the reach of percussion. Consequently, dulness does not extend normally as far to the left of the median line as it does to the right. It is usually about a centimeter less. Neither is the boundary of dulness as definitely shaped on the left. It can easily be understood that any enlargement of the transverse arch may bring its wall nearer to the anterior wall of the chest. The dulness on the left of the manubrium will, consequently, be increased. If, in addition, there is a definitely rounded border to the dulness on the left, we may be sure of some kind of a tumor of the mediastinum. Whether this is an aneurysm, enlarged glands, or a malignant growth, must be determined by other means, but the presence of even a small tumor in the mediastinum can easily be determined by careful percussion. Therefore, we can detect by this means a small aneurysm of the transverse arch as well as of the ascending arch.

Clinically we cannot usually distinguish a sacculated aneurysm from a diffuse aneurysm in this region, nor is the distinction of much practical value—at least in the earlier stages. In a majority of the cases, even when the aneurysm is sacculated, there is also a more or less general aneurysmal enlargement of the surrounding parts of the aortic wall. Moreover, the general dilatation is apt to involve both the ascending and transverse parts of the arch rather than to be confined to one part alone. This is especially likely to be true if there is enlargement of the area of dulness extending to the right and to the left of the sternum. If to the right alone, it suggests the ascending arch; if to the left alone, it probably involves the transverse arch.

If we are to detect cases of aneurysm of the aorta in an early stage, we should make the determination of the width of dulness over the great vessels a routine part of every chest examination, just as we now make the determination of the area of the heart or the examination of the apices of the lungs a matter of routine.

Having determined this area of dulness, we can get important confirmatory evidence of aneurysm by auscultation. A systolic murmur with greatest intensity directly over the dull area is very strong confirmatory evidence, but the absence of a murmur has little significance, and does not exclude aneurysm.

A more important thing to note by auscultation is the transmission of cardiac sounds. The vibrations which correspond to these sounds travel through the column of blood and the arterial wall more

readily than they pass to outside structures. The result is that these vibrations reach the whole wall of the aneurysm with their intensity very little diminished. Therefore, over the area of dulness of an aneurysm we should hear the transmitted heart sounds louder than at parts of the chest outside of this area. This is what actually takes place. We are less likely to have this occur with glands or tumors, even if they lie against the aorta. Therefore, the greater audibility of the heart-sounds over an abnormal area of dulness in the region of the manubrium is very strong evidence that the tumor is an aneurysm.

Another change that may be noted by auscultation in some cases is an accentuated, low-pitched, ringing aortic second sound. Corresponding to this, a diastolic shock is sometimes felt by the hand resting either on the chest-wall or on the bell of the stethoscope. This diastolic shock and the peculiar aortic second sound constitute one of the most characteristic signs of aneurysm of the arch of the aorta. It is usually, however, found in the advanced stages and not in the early stages. However, I have seen it slightly marked in cases of arteriosclerosis with general dilatation of the aorta, even when the amount of dilatation did not cause symptoms and was not sufficient clearly to warrant a diagnosis of aneurysm. Therefore, when slightly marked, this sign may suggest an early stage of aneurysm. When well marked, it is one of the most characteristic signs of aneurysm, but then indicates that the aneurysm is no longer in its early stages. When present, it indicates that the first part of the ascending arch is involved in the dilatation.

Under the head of auscultation we may also consider those signs which may be heard without listening to the chest itself. These signs have to do with the voice and with the respiration. When the aneurysm presses on the recurrent laryngeal nerve certain manifestations may occur, due to imperfect action or to paralysis of the corresponding vocal cord. The effect of this upon the voice may vary from mere "weakness" of the voice, through hoarseness or huskiness, to aphonia. The cough may attract our attention by its peculiar barking or clanging quality. The breathing often has a peculiar quality, due to the wide glottic opening. Pressure on the trachea or bronchus may give a wheezing character to the breathing not unlike that heard in asthma. Usually this comes with inspiration, but it may be with expiration also. Sometimes it has a musical quality not unlike that of a ronchous rale. Any of these breathing sounds, connected with disturbance of the vocal cords, or due to pressure on the air-passages, may occur with an aneurysm that is too small to have reached the surface, and which might not attract attention from the front of the chest.

While considering auscultation as a means to early diagnosis, I have already referred to the possible aid from palpation in detection of the diastolic shock. This sign and the detection of a localized or

general heaving pulsation at the level of the manubrium belong generally, however, to more advanced stages. I mention them because they are so often overlooked. In the early stages, however, palpation is useful in other ways. Its chief aid is in the detection of changes in the pulse and in determining the presence or absence of the tracheal tug. The changes in the pulse may consist either in a loss of strength or of a delay in the time of pulsation of one radial artery as compared with the other. Although this is rarely one of the earliest signs of aneurysm of the arch of the aorta, it frequently comes before there are external signs that are noticeable at the front of the chest. Therefore, the simultaneous examination of both radial pulses should constitute a part of our routine physical examination of patients. We should remember, however, that the difference in the two radial pulses may be due to irregular distribution of the arteries or to pressure somewhere along the course of one of the arteries, and that it is not necessarily due to changes at the arch of the aorta itself.

Another thing that should be noted as a matter of routine in the examination of every patient is the condition of the arterial walls. If we find arteriosclerosis of the radial arteries we should examine the region of the aorta very carefully for possible signs of aneurysm, for arteriosclerosis of the aorta underlies most cases of aneurysm. Particularly should this be done when arteriosclerosis is found in a middle-aged or young individual, for syphilis is thus suggested.

The tracheal tug is a much more valuable sign in early cases. If the trachea is put on the stretch by elevating the chin, a slight tug can be obtained when the aneurysm is still of quite small size. It is conceivable that a mediastinal tumor could give the same phenomenon by taking up space between a normal aorta and the trachea or the left bronchus, but, practically, I have met with little confusion from this source. It should be remembered, however, that retraction of the left lung may draw the left bronchus taut so as to produce the tracheal tug, when the chin is elevated and the trachea is put upon the stretch, even though the aorta is not enlarged. Therefore, it is of importance to examine the condition of the left lung before we give the tracheal tug its full significance in the diagnosis of aneurysm of the arch of the aorta.

Inspection is of little importance in detecting the earliest stages of aneurysm, but by careful inspection we may detect the signs of aneurysm at a much earlier stage than is often the case at present. A slight heaving of the chest wall above the cardiac area is one of the earlier signs. Next to this we are likely to find pulsation in an intercostal space, slight at first, and only to be seen by looking across the chest carefully. We must not forget, however, that visible pulsation to the right of the sternum may be due to displacement of the aorta by deformity of the spine, or to retraction of the lung, which leaves the aorta abnormally uncovered.

An interesting case in my service at the Boston City Hospital during the past winter shows the importance of the proper interpretation of signs seen on inspection in connection with the diagnosis of aneurysm of the aorta.

CASE I.—At the time of the morning visit a man, who had entered the hospital on the previous afternoon, was found sitting up in bed with great distress in breathing, and with a marked cough. The breathing was of a distinctly asthmatic type, and it appeared at first sight to be an ordinary case of asthma. It was noted, however, that with each cough there was a protrusion in the second left intercostal space about over the location of the bronchus. This indicated some obstruction to the exit of air between that point and the trachea, and suggested the existence of bronchiectasis beneath the protrusion. In seeking a cause for the obstruction to the exit of air, pressure on the left bronchus was considered, and percussion and other signs at the manubrium showed the existence of an aneurysm of the transverse arch. Understanding the cause for his dyspnœa, an attempt was made to relieve it by lowering the blood pressure, which was decidedly high. Nitroglycerin was used and proved immediately effective, whereas quite large doses of morphine during the night had failed to give satisfactory relief. The diagnosis was later confirmed by *x*-ray examination.

Pulsation in the episternal notch is another sign that should be looked for on inspection in all cases. We sometimes find in old persons, with wasting of tissues and with slight enlargement of the aorta and great vessels, a pulsation at this place which should not be included under the head of aneurysm. But in other cases any pulsation in the episternal notch needs an explanation and suggests aneurysm. If the pulsation is at the bottom of the notch and its direction is upward, it is likely to indicate aneurysm of the arch. If, however, the pulsation comes from the one side or the other (and it is more common on the right side where the innominate artery lies), then there is enlargement of one of the great branches of the aorta rather than of the aorta itself. It may be noted in passing that palpation is useful in confirming the evidence of pulsation at the episternal notch which is obtained by inspection. By pressing the finger from the episternal notch down between the manubrium and the trachea, such an aneurysm may sometimes be palpated earlier than its pulsation can be detected by inspection. But such aneurysms should be detected at a still earlier stage by careful percussion. While this pulsation in the episternal notch ordinarily indicates an advanced stage of aneurysm of the aorta rather than an early stage, it has been surprising to me to see the number of cases in which the significance of this pulsation has been overlooked. Therefore, it is wise to include this sign when we are considering the detection of aneurysm at an earlier stage.

Of course, all cases of aneurysm of the innominate or of the com-

mon carotid artery do not mean that there is also an aneurysm of the arch of the aorta. But the association of the two is often overlooked. Usually there are not two separate aneurysms, but the visible aneurysm of the branch is the outlying part of a general aneurysmal dilatation which involves both the arch and its branches.

Among the physical signs associated with aneurysm the textbooks generally speak of inequality of the pupils. It seems to me that undue importance has been placed on this sign. When it exists, an aneurysm should be thought of as one of the possible explanations. However, I have seldom found it present except when the diagnosis was evident from other more definite signs; I have often found it absent when I should have expected it to be present; and I recall no case in which it was present in the very early stages. It should be recognized as interesting confirmatory evidence of aneurysm rather than as an essential part of the diagnosis.

Of much more importance in connection with inspection is the detection of evidence of obstruction to the venous circulation. This may involve the superior vena cava or its branches, or the azygos vein. The evidence may consist of localized œdema or of distended veins, or of both. It is suggestive only when confined to the areas of distribution of the above-mentioned veins—namely, the veins of the thorax, upper extremities, neck, and head. If the evidence is confined to a limited area, it furnishes a very definite means of locating the point at which pressure is exerted upon the venous trunks. This sign is of especial importance when the direction of growth of the aneurysm is backward and to the right, and pressure is exerted about the point where the azygos vein empties into the vena cava superior. A small aneurysm at this point may reveal itself more clearly by such evidence of pressure than by other physical signs. Except in this case, however, signs of obstruction to the venous circulation usually belong to advanced stages of aneurysm.

Before considering what symptoms are suggestive of aneurysm of the arch of the aorta, let me give a moment to the history as furnishing possible clues. The commonest predisposing cause is syphilis. Other etiological factors are alcohol, excessive or sudden strain, renal disease, lead poisoning, and gout. With the existence of any of these factors in the history, aneurysm should at least be included among the possible results. We should also remember that while arteriosclerosis is generally an antecedent condition of aneurysm, the most marked condition of arteriosclerosis may exist without giving rise to an aneurysm, and that aneurysms are more common in middle life than in later years—or when active physical exertion is combined with an existing arteriosclerosis. We should remember, on the other hand, that serious arteriosclerosis may exist in the aorta, and yet arteriosclerotic changes in the radial arteries may be extremely slight or may be absent. Therefore, we should not rule out the possibility of aortic aneurysm because we cannot detect arteriosclerosis of the radial arteries.

In considering symptoms in relation to the early diagnosis of aneurysm of the aorta, it should be noted that they are all due in one way or another to abnormal pressure which the aneurysm exerts upon surrounding structures. Their character will depend upon the part which is pressed upon and on the amount of pressure exerted. Symptoms from pressure will be of special importance in the early detection of aneurysm where important structures lie normally in close relation with the aorta, and so related that they cannot be readily pushed aside. Under these conditions *slight* enlargement will cause symptoms. This is the reason why symptoms play so important a part in the detection of an aneurysm of the transverse arch, whereas the ascending arch can push surrounding structures quite a distance before the pressure becomes important.

The deep-seated aneurysms of the descending arch or of the descending thoracic aorta should be mentioned in this connection. Pain (from pressure upon nerves) or difficulty in swallowing (from pressure on the œsophagus) may be the first suggestion of the existence of such an aneurysm. In either case, the aneurysm will usually have first attained considerable size, and we can hardly say that it is in an early stage. However, its deep situation prevents its detection by the ordinary methods of physical examination, and these symptoms are likely to be the earliest available clues. It is in cases of this sort that an *x*-ray examination may give definite information that can be obtained in no other way.

The pain from these deep-seated aneurysms usually comes from pressure backward upon the spinal nerves or their branches. It is usually confined to the distribution of one or two of these nerves, and often is on one side only. The pain constantly recurs at the same place. It may be dull or sharp. A characteristic sign is that it is increased by exertion, because that causes greater pressure by distending the sac. Such pains are often thought to be due to rheumatism or neuralgia. If they are persistent, remain definitely localized, and are increased by exertion, aneurysm should at least be considered. Pressure from other sources might cause the same symptoms. Difficulty in swallowing should at least suggest the possibility of an aneurysm as the cause.

Pain is usually present at some stage of development of aneurysms of the ascending and transverse arch. It is seldom, however, one of the earliest signs, although it may be the first that has attracted the patient's serious attention. It may be localized at the site of the aneurysm, or it may be referred to some of the spinal nerves, as in the case of pain from cardiac disease. It varies from a discomfort to a severe sharp pain. It may be anginoid in type. It is increased by exertion, it is persistent, and is likely to radiate from the mid-chest. Pain, with these characteristics, and without a cardiac condition sufficient to explain it, strongly suggests aneurysm.

Breathlessness, dyspnœa, and sometimes cough, are common

symptoms, due to pressure on the trachea or a bronchus. As they are increased by exertion, it is a very common error to ascribe them to heart trouble. If accompanied by a history of periods of hoarseness or loss of voice, or even "weakness of voice," pressure on the recurrent laryngeal nerve is suggested and aneurysm should be thought of rather than heart trouble. This is even more so if the cough or the respiratory sounds are of the types characteristic of aneurysm. These symptoms of disturbance of breathing are characteristic of aneurysm of the transverse arch, and are often present when the aneurysm is so small as to be made out with difficulty by physical signs.

Of course, breathlessness or dyspnœa on exertion is characteristic of heart trouble as well as of aneurysm. In all such cases the heart should be examined. This is usually done, but too often the examination stops with the heart. Aneurysm should be looked for as well in these cases. Furthermore, aneurysm should be very strongly suspected if the cardiac condition does not give an adequate explanation of the dyspnœa on exertion.

It is not uncommon to have more or less serious heart disease and an aneurysm of the aorta in the same person. In such cases it is not always easy to determine which is responsible for the dyspnœa. In many of these cases the physician, finding a cardiac trouble that seems to account for the dyspnœa, goes no further. He overlooks the aneurysm and treats the case, as a case of broken compensation, with cardiac stimulants like digitalis. This increases the pressure and the distention of the aneurysmal sac, and, consequently, increases the dyspnœa. Then he omits digitalis, and tries nitroglycerin with splendid results, because it lessens the blood pressure. Then he tells of "another case in which digitalis did not agree with the heart, and nitroglycerin acted as a cardiac stimulant." These cases, as well as the high-pressure cardiorenal cases, are the ones that have led the thoughtless physician to look upon nitroglycerin as a cardiac stimulant rather than the vasodilator that it is.

Of course, we do have cases of cardiac disease in which digitalis does not agree, but in these cases it can usually be taken for some little time before this fact is manifest from the cardiac symptoms. If, however, digitalis clearly makes dyspnœa worse as soon as it begins to act, we should suspect the presence of an aneurysm of the aorta, for it offers the most rational explanation of this phenomenon. In 3 cases I have made the provisional diagnosis of aortic aneurysm pressing on the trachea or bronchus from exactly this history—to have it confirmed by physical signs when the examination of the chest was made.

Hemoptysis, caused by the oozing of blood from an aneurysm into the trachea, is not usually regarded as an early sign of aneurysm. Cases of a fatal hemorrhage of this sort from a relatively small aneurysm are reported, however, and I have recently seen an interest-

ing case in which this was the first sign that drew attention to the aneurysm.

CASE II.—A man, aged fifty-four years, had been treated by various physicians for heart disease for two or three years, during which he had complained especially of shortness of breath. He did have mitral stenosis and regurgitation. About one week before February 27, 1907, when he was seen by me in consultation, he began to spit blood. This was regarded as a symptom of passive congestion until he brought up thick, bright blood in considerable amounts. Physical examination showed, besides the cardiac disease, unmistakable signs of aneurysm of the ascending and transverse portions of the arch and of the innominate artery. Dulness over the great vessels extended 4 cm. to the right and 6 cm. to the left of median line. The omission of cardiac stimulants and the use of vasodilators, combined with rest in bed, brought prompt relief of the dyspnœa and a general cessation of the hemoptysis. He improved, and insisted upon going about. During the first three weeks in April he was about outdoors, sometimes walking over half a mile. Then he overexerted himself, and had a return of dyspnœa. On April 30 I saw him again in consultation. He was in extreme distress with orthopnœa, passive congestion of the lungs, liver, and kidneys, and marked œdema of the legs. This time the trouble was clearly from broken cardiac compensation, and not from the aneurysm. Digitalis was ordered in moderate doses, with an increase of nitroglycerin at frequent intervals, in the hope of preventing too great pressure in the aneurysm. Improvement followed.

Before closing the discussion of the diagnosis of these cases I wish to say a few words about the use of the *x*-rays as a help to diagnosis. It certainly is of the greatest aid in many cases. Some cases can be discovered by this means that could not otherwise be recognized, especially aneurysms of the descending arch and of the descending aorta. It usually gives more definite information about aneurysms of the ascending and transverse portions of the arch. When pulsation of the aneurysmal wall can be made out by fluoroscopic examination, it clears up the question whether the tumor is aneurysmal or of another character. When, however, no pulsation is visible, it should not be interpreted to exclude aneurysm.

On the other hand, the *x*-ray examination has disappointed me in sometimes failing to show an aneurysm of the innominate that was plainly evident by percussion. In one case the autopsy confirmed the diagnosis, and in a second case, palpation and inspection clearly showed there must be aneurysm of the innominate. In both cases the fluoroscope alone was used in *x*-ray examination, and apparently the slight increase of shadow due to the small aneurysm was not enough to be recognizable.

A small aneurysm of the transverse arch that gives a shadow little or no wider than that of the spine is extremely difficult to recognize

by *x*-rays. In 3 cases a careful *x*-ray examination failed to show such an aneurysm, yet in each the existence of the aneurysm was later demonstrated by one means or another. In each of these cases I felt warranted in holding to the diagnosis of aneurysm in spite of the failure of *x*-rays to demonstrate it. I believe that in almost all cases a skilful physical examination and a careful consideration of the symptoms will enable the physician to diagnosticate aneurysm of the ascending or the transverse arch, even in the early stages, without the need of appeal to the *x*-rays. The *x*-rays are valuable in these cases, however, as confirmatory evidence, and sometimes in adding definition to the picture. Failure of an *x*-ray examination to reveal an aneurysm does not in all cases exclude its existence.

TREATMENT. It is not my intention to consider treatment in detail, but a few words should be said on the general principles of treatment as affected by the early discovery of these aneurysms. The regulation treatment of the books, involving rest in bed, diet, etc., is directed toward the treatment of the advanced cases that are described. Such treatment is not only unnecessary in the early stages, but it is positively harmful, because it interferes with the maintenance of general good health.

Our problem is essentially the reduction of pressure within the aneurysmal sac. We can do nothing to remove the tissue changes that have already occurred in the arterial wall, and have weakened it. Nature's methods of strengthening this weakened wall are to build up on the outside with connective tissue or to strengthen on the inside with organized blood clot. The latter method is most successful in the sacculated aneurysms with narrow openings from the artery. Relatively few of the aortic aneurysms come under this head, and inasmuch as most of the aortic aneurysms are essentially diffuse in type, very little can be hoped for from the formation of blood clot—especially in the earlier stages. The strengthening of the arterial wall and the development of connective tissue support will be more favorably carried on if we can prevent further stretching of the aneurysmal sac.

In order to prevent further stretching of the wall of the sac we must keep the blood pressure within certain bounds. To do this we must limit the force with which the heart drives the blood, and this is best accomplished by regulating the amount of exertion. In addition, we may have further to lessen the blood pressure by securing peripheral dilatation.

The regulation of the amount of exertion is a matter of judgment in the individual case. If there has been, from occupation or other cause, undue muscular strain, that must be prohibited. But rest in bed is unnecessary in the earlier stages. It has seemed to me that those cases in which I have tried rest in bed have lost by poorer nutrition fully as much as they gained by the avoidance of exertion. I cannot report cures accomplished by the moderate limitation of

exertion—I do not believe a "cure" is possible in many of the cases—but by an intelligent limitation of exertion several patients have lived for years in comfort when otherwise their downward course would have been rapid. One patient lived eight years after the diagnosis was made, performing the ordinary duties of her household, and another patient lived four years, doing light housework and getting much enjoyment out of life by rather extensive travel.

It is essential that the patient should understand what the trouble is, its nature, and the mechanical principles that underlie treatment. Only in this way can we secure intelligent coöperation on his part, and, after all, the success of treatment depends more on his own regulation of his activities than on anything else. Every patient so far, upon learning the real problems of his case, has chosen, as I should do, to live a life of regulated activity rather than enforced rest. They have thus secured comfort and moderate enjoyment, even if they did not live quite as long. Undoubtedly both of the patients above mentioned at times did too much and put an unnecessary strain on the aneurysm, but I am sure neither could have endured greater restriction on the average without an amount of mental unrest that would have defeated the very object of the limitations. We must bear in mind that mental quietude is as important for a tranquil circulation as is limitation of muscular exertion.

Of drugs, the vasodilators are the most useful. The choice and dosage of these drugs are details hardly within the scope of this paper, but I wish to say a word about the iodide of potassium. This drug certainly has won for itself an established place in the treatment of aneurysm, but this has been largely on empirical grounds. It was assumed, but not proved, that this was due to vasodilator action. Some tried to test this by the ordinary blood pressure apparatus, and have argued from their failure that it does not lower the blood pressure.

The thought has occurred to me that certain cases of aneurysm of the transverse arch furnish fully as delicate a means of detecting an increase or diminution of blood pressure as is furnished by the apparatus ordinarily employed. In the yielding wall of such an aneurysm we have the weakest point of the arterial system—the one where change of pressure will be best shown by change in size. Some aneurysms press upon the trachea at just such a degree that a very slight increase of that pressure will produce dyspnœa or cough, while an equally slight lowering of the pressure gives relief. This condition has been clearly present at certain stages of some of the cases under my observation. Other drugs have given relief to these cases in a certain way, and it is accepted as vasodilator action. Is it not at least suggestive that iodide of potassium, though a bit slower, has relieved these cases in exactly the same way? Of course, I recognize the possibility that equally as great relief *might* come in some other way than by lowered blood pressure, but so far no one has been

able to demonstrate what such a method is. I would claim that the conditions of an aneurysm as given above offer as delicate and accurate a means of performing a physiological experiment as does much of the laboratory apparatus. If it is objected that we assume certain conditions and do not see them, I can merely say that the careful, logical consideration of the facts may in some of these cases make the conditions equally as clear and certain as if seen with the eye.

When one can determine his facts well and can can think them out well, he can be sure of detecting aneurysm of the aorta in its earlier stages, and then he can do much to benefit his patient. Nothing more remarkable is needed.

SUMMARY. I may summarize the important points in connection with the physical examination as follows: Include in the routine physical examination the determination by percussion of the area of dulness of the great vessels; if one finds an abnormal area of dulness, determine carefully the conduction of the heart-sounds in this area; include in the routine physical examination the simultaneous examination of both radial pulses; examine for a tracheal tug in any suspicious case; be especially keen for any evidence by inspection or palpation; and, above all, keep aneurysm in mind and remember to examine carefully by all available methods if there is the slightest suggestion in the history or symptoms, or from any physical sign, that an aneurysm may exist. Thorough familiarity with the anatomical relations of the aorta to surrounding structures is of very great value.

I may summarize what has been said of symptoms by noting that, with the exception of the rare cases, in which there is trouble with swallowing, the early symptoms of aneurysm, manifested by pressure, are usually either pain or disturbance with the respiratory apparatus. The latter may come either from pressure on the air passages or from pressure on the recurrent laryngeal nerve. The symptoms frequently simulate those of heart disease, and their true meaning is learned partly by not finding a cardiac condition that will explain the symptoms, and partly by looking for and finding evidence of an aneurysm.

The picture of aortic aneurysm in its earlier stages is not uniform, but varies widely with the position and size of the aneurysm. There are no pathognomonic signs. The most characteristic feature of one case may be entirely lacking in the next one. And yet a careful physical examination and a careful consideration of the physical signs and symptoms should enable us to detect the existence of an aneurysm of the ascending or transverse arch at a very early stage. The *x*-ray examination is of use in the case of aneurysms in these two parts of the aorta as confirmatory evidence, as giving more definite information in some respects, and sometimes (when pulsation is seen) in deciding between an aneurysm and a

solid tumor. The *x*-rays may detect aneurysms of the descending arch and the descending thoracic aorta which cannot be detected by the ordinary methods of physical examination. On the other hand, a negative report of an *x*-ray examination is not absolutely conclusive proof against the existence of an aneurysm.

The detection of an aneurysm of the arch of the aorta requires no greater skill than does the recognition of incipient tuberculosis. It is, therefore, within reach of the general practitioner, if he will give this disease equal consideration with tuberculosis. When discovered early, the treatment is not the same as in the advanced stages. Moderate limitation of exertion and mental quietude are essential, but absolute rest in bed is not necessary. While the disease cannot usually be cured, we can prolong life in comfort. The vasodilators are the most useful drugs so far as medication is demanded.

CARCINOMA OF THE MEDIASTINUM SIMULATING ANEURYSM.

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The following case is reported because of its great interest *per se*, because of the repeated thorough examinations by able men and the indecision or differences of opinion expressed by them, and the ultimate autopsy proof that no one had been able to make a complete diagnosis, the majority of the observers having been of the opinion that the disease was aneurysm of the aorta and no one seriously considering the presence of tuberculosis.

Charles O., aged thirty-three years, came to me February 13, 1905, with the following history: He had always worked hard in mining, beginning to earn his own living at eight years of age by working in the mines in England. At fourteen years of age he went to California and mined and prospected in the west and southwest. From 1895 to 1897 he was mining in South Africa. From 1897 to 1901 he mined in the Klondike, and since 1901 he had worked in the Klondike in summer, returning to the States in winter. He had had no previous illness, except that thirteen years ago he had what was diagnosticated as soft chancre. He was sure he never had any secondary lesions, as rash, sore throat, etc., but in spite of the diagnosis, under medical direction, he took a course of inunctions of mercury, and later a series of baths at Hot Springs, Arkansas. He stated that he had had some enlargement of the veins of his legs and body, beginning seven or eight years ago, but nothing to attract special attention until June, 1904, at which time, while on the steamer on

his way to Alaska, he noticed that his neck was increasing in size, that he could not button his shirt about the throat and was obliged to get collars several sizes larger than usual, and that he felt drowsy most of the time. At Cape Nome he consulted a physician, who told him that he had strained his heart, and must refrain from all active exercise. His neck continued to swell until it measured twenty inches in circumference. He had previously worn a No. 15 collar. He returned to Rhode Island in October, 1904, and during the winter his condition remained unchanged. He had some cough, and at times raised blood-stained sputum. He stated that he had five hemorrhages one night, and was very sick after them. He called a physician who diagnosticated the case as pneumonia, and treated it as such. He was sick about two weeks, and some time later had a second attack of so-called pneumonia, and was confined to the house for about three weeks. Following these attacks he continued to raise blood-stained sputum in small amounts, and at times he raised small clots of dark blood. At about this time he began to have pain, shooting in character, in the right shoulder and down the right arm, and was much troubled by dyspnœa. At this time he came to see me. There was marked swelling of the neck and face, the ears and fingers were cyanotic, and the veins were very much dilated over the whole body. There was a large vein, the size of a lead pencil, on both sides extending from the pectoral region to the groin, the blood current in these veins being downward. The apex beat was slightly displaced to the left. A double murmur was heard at the aorta, the systolic murmur being most audible over the upper part of the sternum and to the right, the diastolic over the middle of the sternum and to the left. There was no measurable expansion of the right side of the chest. Breath sounds were absent over the right lung except on deep breathing, when they were faint. Vocal resonance and fremitus was less marked on the right side, and percussion resonance was but slightly altered. The heart sounds were transmitted quite distinctly to the right as far as the axilla. There were no tubercle bacilli in the sputum. At this time I considered the diagnosis to lie between aneurysm and some form of neoplasm, most probably syphilitic, and gave him iodide of potassium. Under this treatment his condition improved very much, the veins markedly diminished in size, the dyspnœa was much relieved, and the breath sounds became audible over the right lung.

In April, being still in doubt as to the diagnosis, I sent him to see Dr. F. C. Shattuck, who did not commit himself to an exact diagnosis, but was inclined to believe some neolpasm to be the cause of his symptoms. Blood examination was negative. An *x*-ray examination previous to this time showed the heart displaced downward and to the left, with a pulsating mass to the left of the sternum at the arch of the aorta, and the whole right lung somewhat darkened. In May another *x*-ray examination showed that the heart was

not pressed down on the diaphragm so much as previously, and was drawn more to the right; the area of cardiac shadow extended more to the right; the whole right lung was more opaque; and the pulsating mass at the left of the spine remained the same. There was flatness and complete absence of breath sounds over the base of the right lung.

At this time he was again seen by Dr. Shattuck, who still considered it a neoplasm, with the possibility of an aneurysm with a laminated clot between the blood current and the chest wall. At this time, although many of the signs and symptoms were improved, his general condition was poorer than when I first saw him. In June he went to Chicago and at my request saw Dr. R. H. Babcock, and on the advice of friends there also consulted Dr. John B. Murphy. Both of these physicians considered aneurysm of the aorta to be present, and advised his undergoing treatment for this. He returned to Providence and entered the private ward of the Rhode Island Hospital, July 7, 1905, where I followed out the treatment suggested, because, if it were a new growth, no harm would be done, and if it were an aneurysm, he might be benefited. At this time examination differed from the preceding in that the right side of the chest showed some retraction and the left side some bulging, with a slight curvature of the spine toward the left. There were a few small glands palpable on the right side of the neck and one in the right axilla somewhat flattened and about the size of a nickel. The epitrochlear glands could not be palpated. The inguinal glands were palpable but not suggestive of any trouble. The area of cardiac dulness extended 6 cm. to the left of the sternum, 9 cm. to the right, and nearly to the sternal arch. Over the right upper lobe the breath sounds were faint but bronchial in quality. Over the right lower lobe they were absent. Tactile fremitus was absent at the right base and almost absent at the right apex. All sounds were increased over the left lung. There were no rales heard. The blood pressure was 121 mm. Hg. *X*-ray examination differed only in showing increased opacity of the right lung.

The patient was put to bed and kept absolutely quiet; he was given Tuffnell's diet, and a galvanic current was applied through the chest, large electrodes being used, to the point of toleration. Because of stomach symptoms, the diet had to be discontinued, but at no time after this was he able to eat much of anything, nausea being almost constant. Examinations of the sputum for tubercle bacilli were always negative. He failed rapidly, the only change on examination being a gradual extension upward of the dulness in the right chest. The temperature varied between 98.4° and 99°, the pulse between 80 and 100, and the blood pressure was 120 mm.

On August 3 he was seen with me by Dr. Shattuck, who advised giving him greater liberties. The right lung became completely flat, though the breath sounds, bronchial in character, became

audible over the lung (evidently transmitted). Emaciation and loss of strength progressed rapidly, and the enlarged gland in the right axilla gradually increased in size. On September 1 he began to have severe headaches, and a little later his mental condition became erratic. September 14, severe hiccoughing began, which lasted four days. On September 16 he began voiding urine involuntarily. Examination showed flatness over the right upper lobe, with considerable resonance over the right lower lobe, no breath sounds over the upper lobe, and large, moist rales over the front of the right lower lobe. September 18 the hiccoughing ceased, the patient felt much better, and enjoyed his meals, but had difficulty in breathing. Large, moist rales were heard over the front of the chest, marked over the right, less marked over the left. September 19 and 20 he felt fairly well until 8 P.M. in the evening of the 20th, when he began to raise a good deal of mucopurulent material having a very foul odor. Both lungs filled up with mucous rales and his finger-tips became blue. This continued until toward midnight, when he became rapidly worse and died at 2.45 A.M., September 21.

Autopsy. Just to the right of the median line under the clavicle and adherent to the first rib and the upper part of the cartilages of the first and second ribs, is a very hard mass, 6 x 5 cm. thick, firmly attached to the trachea and to the transverse portion of the arch of the aorta. Just below this is another very dense fibrous mass about 2 cm. in diameter, firmly adherent to the descending vena cava and to the right bronchus. The right lung is distended and solidified. In the upper lobe is a large cavity, filled with pus and broken down lung tissue, opening into the right bronchus. There are similar areas of softening in the right lower lobe. The bronchioles are filled with a creamy material. The lung is so adherent to the chest wall that it can be removed only by tearing. On the outer surface of the lower lobe is an area 2 x 3 cm. in diameter, where the pleura is nearly 1 cm. thick, yellowish in color, and quite firm. The kidneys and liver are dark colored and slightly congested, but otherwise normal. The left lung is normal. The head was not opened.

Microscopic Examination. Tissues were taken from the lung and mediastinal growth and examined by Dr. Frank T. Fulton, pathologist to the Rhode Island Hospital, who reported as follows: Sections from the mediastinal tumor were made up of dense, fibrous stroma, in which were scattered areas of epithelial cells. These areas of cells vary very much in their size and are entirely irregular. They, as a rule, are retracted from the surrounding connective tissue stroma. The cells forming the outer border of these groups of cells are arranged in a fairly definite irregular layer, but the other cells, the cells within, are very irregular in their arrangement, although fairly regular in size. The cells are rather large, and have a pale nucleus and a moderate amount of protoplasm. The nucleoli

are fairly prominent. Mitotic figures are fairy abundant. The central part of most of these cell areas is completely necrotic, in some instances, however, the form of the cells is still preserved, but their ability to take the differential stains is lost. In other of the groups are seen areas closely resembling pearly bodies, that is, made up of cells which are slightly cornified. There are, however, no very pronounced pearly bodies. The whole appearance of the growth is such as to lead one to believe that it originated from epithelium of the epidermal form.

Sections from the lung show the outer surface to be covered by a thick layer of fibrin in which there are a few scattered groups of leukocytes. The lung itself in the part from which the selections are taken has lost its normal appearance. There are very few alveoli which even approach normal, and these are filled with mononuclear cells and leukocytes. In some places there is granulation tissue, in which are perfectly typical small tubercles showing the necrotic centre with the surrounding lymphoid, epithelioid, and giant cells.

Anatomical Diagnosis. Epidermoid carcinoma; pulmonary tuberculosis.

Thus we had, in this case, a sudden onset of symptoms in a man who had undergone exertion and privation of almost every sort, including work in the mines when a boy, packing over the Chilkoot Pass, starvation, and making money and losing it again; the chief symptoms were a remarkable dilatation of the veins of the whole body, but especially of the head and neck, a double murmur at the aortic orifice, slight displacement of the heart, a pulsating mass to the left of the spine at the arch of the aorta, the expectorating of a moderate amount of blood, and some pain radiating from the heart. These symptoms were certainly in harmony with aneurysm of the aorta. Against this was the lack of any bruit or expansile pulsation, and there was no laryngeal paralysis or tracheal tug. In addition to the absence of bruit and expansile pulsation was the relief given by iodide of potassium, the subsequent change in the position of the heart, and the appearance of enlarged glands in the axilla and above the clavicle. I could not at any time consider that an aneurysm accounted for these things.

For a number of weeks before death I believed the condition to be malignant disease, but I believed that the involvement of the right lung also was of cancerous nature. I was very much surprised when the lung was demonstrated to be tuberculous, especially considering the fact that until about forty-eight hours before death the patient's temperature was rarely above 99°, and at no time were tubercle bacilli present in the sputum.

Judging from the character of the tissues and the symptoms presented, the older of the growths pressed on the vena cava.

On section, this growth cut almost like pure fibrous tissue. Just when the tuberculosis of the lung started it is difficult to say, but it evidently had nothing to do with the symptoms or signs presenting themselves until the later part of his illness, though the firmness of the pleural adhesions indicated that they were of long standing and may have been of tuberculous origin, and a latent tuberculosis of the pleura may have developed into an active tuberculosis of the lung as a result of lessened vitality from non-aëration.

I believed that the signs at the base of the right lung were due to the pressure of some growth upon the right bronchus, and I still believe this to have been the case. That the blood expectorated was due to tuberculosis I do not believe, because no bacilli could be found in the sputum, because the signs were those of a collapsed rather than of a solidified lung, and because the *x*-rays showed a diffused cloudiness rather than a localized opacity, as one would expect if tuberculosis were sufficiently advanced to destroy the walls of the bloodvessels so as to cause a hemorrhage.

There was, thus, a carcinomatous growth occluding by pressure the superior vena cava, passive congestion and dilatation of the veins of the whole body, this dilatation being very marked over the upper half of the body, he rupture of some bloodvessel in the lung as a result of the passive congestion, pressure of the carcinomatous growth on the right bronchus, a secondary carcinomatous growth under the upper part of the sternum displacing the aorta to the left, involvement of the right axillary and cervical lymph nodes, infection with tubercle bacilli of the partially aërated lung, gradual extension of the tuberculosis, necrosis of practically the whole lung, and the breaking of this necrotic material into a bronchial tube causing death, the whole so closely simulating aneurysm of the arch of the aorta that but two of the physicians who saw the patient were willing to consider it anything except aneurysm.

CARDIAC ARRHYTHMIA DUE TO EXTRA-SYSTOLES ORIGINATING IN THE BUNDLE OF HIS.

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The following case illustrates a condition of which very few examples have been reported; in fact, its occurrence is only beginning to be recognized.

The patient, H. B., entered the Montreal General Hospital in December, 1906, complaining of shortness of breath of six months' duration and of cough for the preceding month. He gave a history of the free use of tobacco and of a severe strain in lifting a piece of machinery just before the onset of the symptoms. There was no history of rheumatism or syphilis. His condition on admission was as follows: A well-nourished man, aged forty-one years; skin and mucous membranes cyanotic; cough, with profuse mucopurulent expectoration. Cold extremities. Marked dyspnœa on

FIG. 1.—Radial pulse tracing. Time in $\frac{1}{5}$ seconds.

exertion. Pulse small, easily compressible, and irregular (Fig. 1). Blood pressure, 98. Heart impulse weak. Area of cardiac dulness uniformly increased to a moderate extent. Sounds clear, but faint; no murmurs. A premature systole could be heard every second or third beat. There were evidences of pulmonary œdema at both bases. Under rest in bed, with administration of strychnine, the pulmonary œdema cleared up and the area of cardiac dulness diminished, but the rhythm of the heart did not show any material improvement. A number of tracings were taken with a small portable

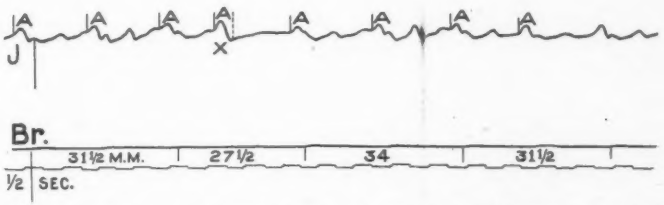

FIG. 2.—Tracing from the internal jugular vein and the brachial artery. Time in ½ seconds. Pulse intervals (bigeminal) are measured in millimeters. Time relations cannot be explained by ordinary ventricular extra-systoles. The high auricular wave at × cannot be explained by auricular extra-systoles.

kymograph from the internal jugular vein and the brachial artery. Typical examples are shown in Fig. 2. A number of extra-systoles were recorded, but they all agreed in essential points with that represented in Fig. 2. The waves marked *A* in the jugular pulse are caused by the auricular systole. In that under which a cross is placed the ventricle is contracting at the same time, as shown by the unusual height of this wave and by the absence of the systolic wave and ventricular collapse from their ordinary positions. The brachial pulse corresponding to this heart-beat is not seen, being too weak to record itself.

The combined wave *X* occurs before either the ventricular or auricular systole is due. The auricular wave is not due, as estimated by the two preceding pulse intervals, until the point marked by the dotted vertical line. We, therefore, have the auricle and ventricle contracting simultaneously before either is due. We might explain their contracting together by a ventricular extra-systole, but this would not explain the premature contraction of the auricle. We can exclude ordinary ventricular extra-systole, moreover, from the fact that the first compensatory pause is very much too short. The two normal pulse intervals preceding the extra-systole together measure 31.5 mm. (in the original tracing the figure is reduced in size), the short and long interval separated by the extra-systole together measure only 27.5 mm., and the next two 34 mm. Compensation, then, is ultimately nearly perfect, but is spread over three pulse intervals, which excludes ordinary ventricular extra-systoles. We can explain the facts on the hypothesis that there is an extra stimulus acting on the auriculoventricular bundle of His. This travels down to the ventricle and up to the auricle, bringing about a simultaneous contraction of the two chambers. This hypothesis is the only one that seems to meet the case.

James Mackenzie[1] and K. F. Wenckebach[2] have drawn attention to the possibility of extra-systoles starting in the auriculoventricular bundle, and H. E. Hering[3] claims that it is a comparatively frequent occurrence. If this is the case, it is important that the means of recognizing it be more widely known, so that in time we may learn something of its etiology and of its bearing on diagnosis.

SOME OF THE REASONS WHY CHOLECYSTECTOMY SHOULD NOT BE PERFORMED AS FREQUENTLY AS IS ADVOCATED BY MANY SURGEONS.[1]

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Before speaking of cholecystectomy it may be well to recall briefly the physiology of the gall-bladder and the bearing that interference with its functions may have upon the subsequent condition of the patient. The functions of the gall-bladder are in brief these: It acts as a reservoir for bile, and as a tension bulb for the biliary

[1] Observations on the Inception of the Rhythm of the Heart by the Ventricles, Brit. Med. Jour., 1904, i, 529–536.
[2] Archiv für Physiolog., Leipz., 1905.
[3] XXIII Congress for Internal Medicine, Munich, 1906.
[1] Read at a meeting of the Southern Surgical and Gynecological Association, New Orleans, La., December 18, 1907.

passages through the medium of which the *vis a tergo* of the bile stream is equalized. The force of the current of the bile stream is best illustrated by what occurs after the removal of a normal gall-bladder, when, unless the cystic duct has been tied very securely and with a ligature that will not be absorbed too quickly, the *vis a tergo* of the bile current demonstrates itself by opening the stump of the cystic duct, with resulting escape of bile. I have had this happen in my earlier experience before I appreciated the importance of this knowledge of the force of the back-flow of the bile.

While I am aware that patients who have had their gall-bladders removed may remain perfectly well, this does not demonstrate that the gall-bladder does not serve a useful purpose; the judicious surgeon—the surgeon who regards Nature and assists her as far as he can—will not ablate a gall-bladder which is still functionally active or which may later become functionally active. The fact that a diverticulum from the stump of the cystic duct or along the course of the common duct occasionally develops after the removal of the gall-bladder, is sufficient proof to me that the gall-bladder was placed where it is for a useful purpose. On the other hand, I have never seen this result follow ablation of a gall-bladder which had long ceased to functionate; evidently in such cases Nature has accommodated herself by slow degrees to the absence of a reservoir for the bile.

The gall-bladder is more active as a reservoir during the longest interval between eating, which is during the night; and it is for this reason that people with normally functionating gall-bladders sleep better, other things being equal, than do those who are not so fortunate. I have sometimes thought that, perhaps, the reason that persons with gall-bladder affections sleep better after a late supper than might be expected of persons in their condition, is that the process of digestion keeps the gall-bladder more or less empty of bile, and that thus the presence of the lesser evil procures less disturbed sleep.

From the standpoint of subsequent pathology the gall-bladder is surely a most useful organ by way of which to drain in certain septic conditions of the liver and biliary passages. It must be admitted that drainage is the essential feature in all truly infectious diseases of the biliary passages; and as the use of the gall-bladder for this purpose, whenever possible, is safer for the ordinary surgeon than either of the deeper and more dangerous operations, namely, drainage of the common duct or of the hepaticus, the gall-bladder should be preserved whenever possible.

When chronic pancreatitis occurs after removal of the gall-bladder, prolonged drainage is indicated; and since, even if the common duct be obstructed, it is very exceptional to meet with such a degree of dilatation as will permit of a choledocho-enterostomy, the surgeon is hard put to it to maintain drainage for a sufficiently long time. If, however, the gall-bladder has not been removed, a cholecysto-

enterostomy will solve the problem by giving not only a shorter convalescence, but a more certain recovery due to the permanent drainage.

In the absence of the gall-bladder, and in the presence of an hepatic emergency, the surgeon will be obliged to drain the common duct or the hepatic duct itself. Each of these procedures is an operation which may tax the skill of the expert abdominal surgeon; and if so, What must they be for surgeons of limited experience in this line of work? The gall-bladder and cystic duct, anatomically, hold the same relations to the common duct that the cecum and its anterior longitudinal, muscular band do to the appendix; therefore the presence of the gall-bladder and the cystic duct is the white-signal light to the surgeon who has to expose and drain the common duct in the absence of a foreign body—which latter will serve to locate the duct. For the above reasons, as well as those I shall presently mention, we should feel keenly our responsibility when reaching a decision to do a cholecystectomy or a cholecystostomy.

When a gall-bladder is removed in the presence of infection of the bile passages, drainage should be established; when it is possible the stump of the cysticus should be drained; and if this cannot be done drainage of the hepaticus by means of the choledochus should be done, in all cases a rubber tube being introduced.

Some surgeons assert that after cholecystectomy sufficient drainage takes place by means of the choledochus into the duodenum, but it is much wiser to drain outside of the abdomen than to allow the infectious bile to flow into the duodenum, from which, as well as from the bowel further along, the toxic elements in the bile may be taken up by the tributaries of the portal vein, and the hepatic cells thus be exposed to the risk of re-infection. To me it is incomprehensible that surgeons should regard drainage of the hepatic duct through the common duct into the duodenum as sufficient in cases of cholangitis. To drain one infected organ into another—the duodenum—I consider unsurgical in the extreme. In cases of severe pyelonephritis we are not satisfied with the drainage afforded through the ureter into a bladder which was probably the original seat of the disease; we perform nephrotomy and drain the kidney directly into the loin. So when the liver is infected and there is suppurative cholangitis, the only chance of safety lies in direct hepatic drainage through the laparotomy wound.

When in doubt I say drain rather than remove a gall-bladder which may recover itself; better do this even at the expense of a fistula (mucous) which will call for a second operation, than take out a gall-bladder which may again normally functionate. Better, I say, do two operations in the attempt to save a gall-bladder that may recover itself, and in not a few of these instances save a human life at the same time, than do an operation which will certainly sacrifice the one if not both.

That the mortality from drainage of the gall-bladder is less than that which attends its removal, we know. We must consider, moreover, the remote as well as the immediate mortality following cholecystectomy: this includes peritonitis from spreading infection, from hemorrhage which kills within two or three days, and the small bleeding responsible for a still later peritonitis—to say nothing of the additional shock to the patient by the latter operation, which entails more time and longer intraperitoneal manipulation; and these are very serious matters in many gall-bladder cases. This prolongation of the operation too often throws an additional burden upon already toxic kidneys as well as upon other emunctories which they can ill afford. I have no hesitancy in proclaiming my partiality for the simpler of the two operations; notwithstanding the fact that my views are at variance with those of many operators, my individual experience has taught me the wisdom of taking this stand.

That the question of postoperative adhesions is a matter of more moment after cholecystectomy, I know from repeated experiences in operating for the relief of symptoms thus caused. It is only necessary to recall the anatomical relations of the viscera in the immediate neighborhood of the under surface of the liver, and our utter inability, in many instances, thoroughly to cover all of the exposed surfaces following the ablation of the gall-bladder, to arrive at a just appreciation of the crippling power of postoperative adhesions in this region.

Bland-Sutton in a recent article on the "Indications for the Performance of Cholecystectomy," makes the assertion that "Observations in the dead house show that disease destroys it (the gall-bladder) very often without even causing inconvenience to the individual." This is a rather brave assertion, particularly when the dead cannot speak.

The occurrence of biliary colic is very rare after the removal of stones and drainage. What is not very uncommon, is colic the result of adhesions; this, if it occurs, calls for opening of the abdomen and release of the adhesions. The recurrence of stone as an objection to cholecystostomy may practically be dismissed. The chief objection to cholecystostomy is the occurrence of fistula; with our present methods, however, this is much less frequent than formerly when we attached the gall-bladder to the abdominal wall.

Fistulæ are of two kinds: mucous and biliary; the former is the result of obstruction of the cystic duct; in the latter the cystic duct is patulous as a matter of course, otherwise we should not have a free flow of bile. The former I admit will require, in practically all instances, cholecystectomy; the latter not. I have been able to close most of these biliary fistulæ by freeing the fundus of the gall-bladder at the point of attachment, and turning in the margins of the opening and suturing; occasionally I have found it necessary to drain the gall-bladder (without attaching it anew to the abdominal

wall); and in a few instances I have made a cholecystoduodenostomy. Rarely, if ever, has it been necessary to do a cholecystectomy. The gall-bladder should not be removed unless we feel that by so doing we are taking away a menace to the future health and comfort of our patient.

Silk should never be used in the gall-bladder for repair or for the retention of a drainage tube; even chromicized catgut ligatures may act in the same way as silk, forming a subsequent nidus for gallstones.

In hydrops of the gall-bladder cholecystectomy is practically always required, since obliteration of the cystic duct renders the gall-bladder not only useless, but a continued menace from the liability to re-infection and even rupture. The only exception I would make to this rule is in obstruction of the cystic duct by stone if of recent standing; if the gall-bladder shows but little macroscopic evidence of disease, and upon dislodgement of the stone bile flows immediately into the gall-bladder, it may be wise to leave the gall-bladder.

Cholecystectomy should be done in cancer or other new-growths of the gall-bladder when they are limited to the gall-bladder; and in calcareous degeneration or in fibrosis of the gall-bladder.

Traumatism of the abdominal wall with rupture of the gall-bladder has occurred twice in my experience; in neither case was it necessary to do more than drain.

Chronic empyema calls for cholecystectomy, while in acute empyema drainage, may suffice. Gangrene of the gall-bladder naturally requires its excision, and the same is true of perforation, since this last condition occurs most frequently when the gall-bladder wall is extensively diseased. In the presence of gangrene of the gall-bladder when there are severe constitutional symptoms, it may be wise to leave the gall-bladder temporarily; this has been done, and removal accomplished at a subsequent period, with good results. I have seen many acute empyemas recover after drainage only.

When the fundus of the gall-bladder only is diseased it should be resected, and the remaining part of the gall-bladder drained.

Ablation of a gall-bladder, the seat of stones, but otherwise healthy, is indicated when many small stones, all of which cannot be removed, are embedded in the mucous membrane. It will readily be understood that if even a single stone remains behind, we take chances on subsequent stone formation.

The only way of determining a patulous cystic duct, before opening the gall-bladder, is by palpating the duct for induration of its coats or wall (after division of the peritoneal covering), to see if it is kinked or pouched, and compressing of the gall-bladder, which, if the duct is open, should rapidly empty: the latter method is by far the more reliable. The gall-bladder should not be removed unless

the cystic duct is permanently and irremediably occluded, and the walls seriously diseased. With few exceptions, when the duct is patulous, the gall-bladder should not be removed in acute catarrhal calculous or non-calculous cholecystitis; nor even always in acute suppurative calculous or non-calculous cholecystitis. In but comparatively few cases of chronic calculous or non-calculous cholecystitis is ablation indicated, unless hydrops, fibrosis, or calcification of the wall of the organ exists. I do not believe that the removal of the gall-bladder prevents absolutely the recurrence of stone, for we know that stones, though rarely, are found in the smaller hepatic ducts and in the common duct.

In gall-bladder work, as well as in other departments of surgery, hard and fast rules cannot always be followed. Whether the gall-bladder shall be taken out or not must sometimes be decided not only by the condition of the organ itself, but also by the condition of the patient. While cholecystectomy may be proper when the gall-bladder alone is involved, it may not be proper in the presence of associated disease of the bile ducts.

This question of cholecystectomy, pro and con, appeals to me strongly. From what I read and hear, I fear that removal of the gall-bladder is done too frequently; and I feel that the influence of the prevalent teaching in favor of cholecystectomy upon those who have not had sufficiently large experience to decide for themselves for or against removal, will be bad indeed. Personally I can honestly say that the more gall-bladder surgery I do, the less inclined I am to remove the gall-bladder.

PERFORATION OF THE GALL-BLADDER DURING TYPHOID FEVER; CHOLECYSTECTOMY; RECOVERY.[1]

WITH AN ANALYSIS OF TWENTY-ONE OPERATIONS ON THE GALL-BLADDER DURING TYPHOID FEVER.

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Although inflammation of the gall-bladder is frequently observed in typhoid fever, and though numerous cases of post-typhoid cholecystitis have been reported, yet operations for perforation of the gall-bladder during the course of the disease are sufficiently rare to make them worthy of record. For the privilege of reporting two cases, and of operating on the second patient, I am indebted to Dr. G. G. Davis, in whose service at the Episcopal Hospital they occurred.

[1] Read at a meeting of the College of Physicians of Philadelphia, November 6, 1907.

CASE I.—*Perforation of the gall-bladder on the thirty-second day of typhoid fever; exploratory laparotomy; death in twenty hours.*

Clarence S., aged twelve years, was admitted January 26, 1907, to the medical wards of the Episcopal Hospital, under the care of Dr. A. A. Stevens. He had been ailing for about three weeks, but had been confined to his bed at home for the last week only, complaining of pain in the abdomen, nausea, vomiting, headache, and diarrhœa. There had been no epistaxis. On admission he presented a typical picture of a severe case of typhoid fever in the fourth week: face flushed, pupils dilated, tongue coated and dry, lips dry. Slight bronchitis. Heart normal. Abdomen soft; spleen enlarged and palpable; rose spots present. The Widal test was positive, and the leukocytes numbered 10,800. His temperature was 101.8° F.; pulse, 104; respirations, 24.

January 28. Quite delirious; moaning all the time in his sleep.

February 3. Still has mild delirium. Temperature has varied little, ranging from just below 103° F. to over 104° F. His pulse rate has varied from 120 to 130; and his respirations have been about 24 per minute.

5*th*. (Thirty-second day of disease). At 10 A.M. was found to be in pain; had not complained of sudden onset of pain. He now has pain in right side of abdomen. Temperature, 104.4° F.; pulse, 132; respirations, 24. Leukocytes, 10,200. There is some rigidity of the right rectus muscle; the most tender spot is over the belly of right rectus, on level of umbilicus. No appreciable tenderness on left side. No distention.

11 A.M. Pain, tenderness, and rigidity considerably increased. The abdomen moves very little with respiration. The leukocyte count has fallen from 10,200 at 10 A.M., to 6600 at 11 A.M. 1 P.M. Pain, tenderness, and rigidity still more increased; also more distention. Tenderness is no longer so sharply localized, but has spread over entire abdomen; yet the most tender area is still on right side. 2 P.M. Temperature, 102.4° F.; pulse, 120; respirations, 36. Leukocytes, 6200. A study of the temperature chart shows that there had been a steady rise of temperature, beginning the morning of February 3 (forty-eight hours ago), from 101.8° to 104.4° F., and that on the occurrence of perforation this morning there was a fall of two degrees (to 102.4° F.) within three or four hours. There has been practically no change in the pulse rate. The leukocyte count fell within four hours from 10,200 to 6200. A diagnosis of probable intestinal perforation was made. 3 P.M. Laparotomy (ether) by Dr. G. G. Davis, five hours after the occurrence of perforation. A transverse incision was made in the right iliac region, at the level of the anterior superior iliac spine, splitting the anterior and posterior sheaths of the rectus and the transverse abdominal muscles. Considerable bile-stained fluid was found on the gauze sponges inserted among the intestines. The entire small

intestine and about six inches of the ascending colon were explored, but no perforation was found. All the intestines were markedly injected and inflamed. On account of the patient's critical condition, no further attempt was made to find the lesion causing the peritonitis. A rubber tube and iodoform gauze drain were introduced and the wound partially closed.

6*th*. The patient died at 11 A.M., twenty hours after operation. Chemical examination of the fluid removed at operation showed the usual reactions for bile. Postmortem examination through the wound of operation showed general peritonitis, with a perforation of the gall-bladder.

CASE II.—*Perforation of the gall-bladder on the forty-second day of typhoid fever; cholecystectomy; recovery.*

John S., aged twenty years, was admitted February 15, 1907, to the medical wards of the Episcopal Hospital, under the care of Dr. A. A. Stevens. His chief complaint was diarrhœa and weakness. His family history was negative, and he had never been ill before. His present illness began two weeks before admission, with headache, malaise, anorexia, cough, diarrhœa, and general weakness. There had been no epistaxis. He had not been in bed constantly before admission.

On admission his temperature was 104° F.; pulse, 120; respirations, 24. His face was flushed, his pupils normal, his tongue coated and moist, and his throat clear. His lungs were clear, and his heart was normal. The abdomen showed slight distention and tenseness; no rigidity; no gurgling; some general tenderness. Spleen very slightly enlarged to percussion. There were a few typical rose spots on the abdomen. The Widal test was positive, and the leukocytes numbered 8000.

His temperature pursued a rather irregular course, falling considerably after each bath, then rising again, but showing a gradual tendency to subside. It became normal about the thirty-seventh day of the disease, but again began to rise irregularly on the thirty-ninth and fortieth days. For a week he had not been tubbed. His bowels had been constipated, but were opened occasionally by enemas. His pulse varied from 68 to 80.

March 14. (Forty-first day of disease.) His temperature shot up to 103.2° F. at 4 A.M., and he was tubbed. At 8 A.M. his temperature was 104° F., pulse, 120; and he was again tubbed. He now vomited some greenish fluid. At 10 A.M. he complained of not feeling so well as on previous mornings. He complained of no pain anywhere. On deep palpation below the right costal border there was slight though distinct tenderness. No mass palpable. Apparently no distinct rigidity. His general condition was good. The leukocytes numbered 24,400. At noon, temperature, 104.8° F. No bath. Ordered calomel, grain $\frac{1}{4}$, hourly for six doses, to be followed by half an ounce of Epsom salt. At 4 P.M., temperature, 104.2° F.

No bath. At 8 P.M., temperature, 103.6 F. No bath. At 11.30 P.M. it is noted that both local and general conditions had remained about the same during day. Patient had vomited all medicines during the day. Probably the tenderness below the right costal border was slightly increased. Temperature, 104.4° F.; pulse, 116; respirations, 28.

15*th*. (Forty-second day of disease.) 4 A.M., temperature, 100.2° F.; pulse, 136; respirations, 28. Pain and tenderness are now severe in right upper abdominal quadrant. He has vomited some more green fluid. The skin is somewhat leaky. The pulse is rapid, but of good volume. Entire right half of abdomen is very rigid, but practically all pain and tenderness are above the level of umbilicus. There is no distention, nor is there dulness in the flanks.

A diagnosis of perforation of the gall-bladder was made by the resident physician, Dr. von Buddenbrock, and I was sent for.

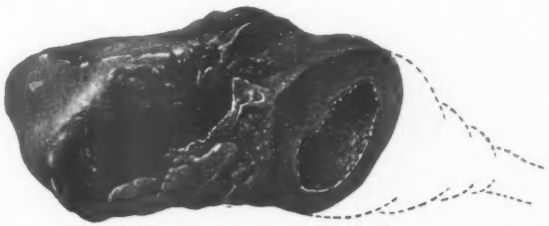
When I first saw the patient he looked like a man of forty years. It was surprising to be told he was only twenty years of age. His expression was characteristic—not the Hippocratic facies, but rather a general weakening of all the features, a change which has been particularly alluded to by Dr. Harte.

With the history of a rapid rise of the temperature from nearly normal almost to 105° F., followed by abdominal pain, tenderness, and rigidity, the existence of intra-abdominal mischief was sufficiently evident. The localization of the symptoms to the right upper abdominal quadrant, and the memory of the perforated gall-bladder observed only a few weeks previously, strongly suggested that the gall-bladder was the seat of trouble in this patient. Following this train of symptoms, which had rapidly developed within the last twenty-four hours, came the sudden drop of more than four degrees in the temperature within three hours, with the coincident quickening of the pulse from 110 to 136 and the occurrence of sweating. With such a history and train of symptoms, the diagnosis of perforation of the gall-bladder seemed justified.

At 8 A.M., four hours after the occurrence of perforation, I operated on the patient under ether anesthesia. A small exploratory incision was made through the outer border of the right rectus muscle, splitting its fibers, above the level of the umbilicus. There was a little free serum, but there were no adhesions to the parietal peritoneum. As the transverse colon was seen to be adherent above to the liver, the incision was extended upward, its total length being four inches. The upper abdominal region was then carefully isolated by gauze packs, and the adhesions, which were light and evidently of quite recent formation, were cautiously ruptured. There at once welled up from above the transverse colon some more serous fluid, which was quickly followed by four or five ounces of thick, inodorous, milky pus. This pus, which seemed to come

from the under surface of the liver, was rapidly sponged away. (A culture made during the operation gave a pure growth of typhoid bacilli.)[2] The pylorus and duodenum were then recognized, as well as the round ligament of the liver. Then the gall-bladder was found, further to the patient's right, buried in rather dense adhesions between the liver and the colon. There was a large and ragged perforation, measuring $1\frac{1}{4}$ x $1\frac{1}{2}$ inches, in its lower and inner wall, not involving either fundus or neck, and accounting by its size for the collapsed condition of the gall-bladder. The gall-bladder was enucleated; its neck was ligated with chromic catgut, and it was cut away (see figure). A rubber tube and two gauze packs were inserted as drains; the isolating packs were removed, and the abdominal incision partly closed with through and through silkworm-gut sutures.

The operation lasted twenty-three minutes, and the patient was in a very precarious condition at its conclusion. A wet dressing of alcohol and salt solution was applied over the whole abdomen,



Gall-bladder removed by cholecystectomy, showing typhoid perforation. (Natural size.)

and in the manner of a poultice was covered with waxed paper and absorbent cotton. Throughout the operation the patient had been surrounded by hot-water bottles, and every effort was made to prevent chilling him.

The patient was given one-thirtieth grain of strychnine sulphate and ten minims of the tincture of digitalis, every fourth hour, hypodermically. He also received six ounces of hot saline solution, with four ounces of peptonized milk, every fourth hour by enema. He was given one hypodermic injection of one-eighth grain of morphine sulphate.

16*th*. There was some bilious vomiting, but this was arrested by washing out the stomach. His head was clear, and he felt well except for tenderness over the wound.

[2] My thanks are due to Dr. D. L. Despard, assistant pathologist to the Episcopal Hospital for his very painstaking and persistent search for micro-organisms in sections cut from the margins of the perforation in the gall bladder. In spite of the use of varied stains and the examination of numerous sections, he was unable to demonstrate the presence of the typhoid bacillus in the tissues.

17*th*. The dressings were bile-stained (the third day); fluids were given by mouth; also one-half ounce of whisky every sixth hour.

20*th*. Five days after the operation the patient was lying on his right side, and considered himself convalescent. Gastric lavage was again employed for bilious vomiting. The drainage tube was removed, and the gauze on the gastric side was loosened.

21*st*. Six days after the operation the temperature reached normal. "The patient lies on right or left side. There has been no more vomiting."

With the exception of one rise of temperature to 101° F., recovery was uneventful. The last gauze was withdrawn, and feeding on soft diet was begun, on the ninth day after operation No pus discharged from the wound after the first week, and the discharge of bile gradually lessened. In the fourth week after operation the temperature rose for a couple of days to 103° F., owing to the development of an acute otitis media; and in the fifth week a furuncle on the leg caused a rise to 103° F.; but finally convalescence was assured. The sinus ceased to discharge in the fifth week, and the patient was sent home, well, May 15, 1907, just two months after the operation.

The successful result in this case may be attributed to several factors. Among causes which may be said to have predisposed to his recovery are: (1) The stage of the disease at which perforation occurred, when the height of the fever was past; (2) the existence of a pure typhoid infection, uncontaminated by the more deadly colon bacillus or by the streptococcus; (3) the youth of the patient —only twenty years—and his good constitution; and (4) the short period—four hours—that elapsed between perforation and operation. The excellent tissue reaction shown by the patient, evidenced by the leukocytosis of 24,400, and by the promptitude with which protective adhesions were formed, must also be considered. As exciting causes should perhaps be mentioned the fact that the gall-bladder was excised, and that such care was taken to prevent diffusion of the infection below the transverse colon. Two months in the hospital after either a cholecystectomy or an attack of typhoid fever would be an abnormally long sojourn, but when both occur simultaneously in the same patient, the rate at which strength is regained is exceptionally slow.

This patient was seen again in October, 1907. He was in excellent health, had been at his work as a paper-cutter all summer, and felt no inconvenience from the operation. The scar had contracted to a length of less than three inches; it was firm and unyielding, and there was no impulse on coughing.

In connection with these 2 cases from the Episcopal Hospital, I have analyzed the reports of 19 other operations done during the course of typhoid fever for lesions of the gall-bladder. A number

of operations done after complete recovery from the disease, for post-typhoid affections of the gall-bladder, have not been included. Such operations have been recorded by Gibbon,[3] Harte,[4] Halsted,[5] Deaver,[6] Martin, Frazier[7] and other surgeons. A case reported by Dr. H. B. Allyn,[8] in which operation was done by Da Costa for perforation of the colon into an adherent gall-bladder, was included in the tables I published some years since, in collaboration with Dr. Harte;[9] it is not included in the present table because it was thought by Dr. Allyn that the perforation originated in the colon, not in the gall-bladder.

Taking, then, only those 21 operations done before the patients recovered from typhoid fever, we have a record of 8 recoveries and 13 deaths, or a mortality rate of 61.9 per cent. In 4 cases, all fatal, the operation was abandoned before the gall-bladder lesion was found. Should it seem proper to anyone to exclude these cases, it would leave a total of 17 completed operations, with 8 recoveries and 9 deaths, or a mortality rate of less than 53 per cent. Compared with operations for intestinal perforations during typhoid fever, which give an average mortality, in collected cases, of from 75 to 80 per cent., operations on the gall-bladder during typhoid fever may seem almost benign; but their real gravity is apparent when they are compared with similar operations on patients without constitutional disease. In these the mortality varies from 3 to 10 per cent.

The operations employed in the present series of cases may be seen in the following table:

Operation.	Cases.	Recovered.	Died.	Mortality. Per cent.
Aspiration of gall-bladder through unopened abdominal wall	1	1	0	.....
Cholecystendysis	2	1	1	50.00
Cholecystotomy and drainage	9	4	5	55.55
Cholelithotomy	2	0	2	100.00
Cholecystectomy	3	2	1	33.33
Operation abandoned without finding lesion	4	0	4	100.00
Total	21	8	13	61.90

The number of cases involved is, of course, too small for any definite conclusions to be drawn from a statistical study; but it may be said, in brief, that this complication seems to be most frequent in females between twenty and forty years of age, and during the second or third week of the disease.

[3] Annals of Surgery, 1901, xxxiii, 70.
[4] Ibid., 71.
[5] Trans. Assoc. Amer. Phys., 1897, xii, 390, Case 2.
[6] A. O. J. Kelly. AMER. JOUR. MED. SCI., 1906, cxxxii, 459, Case 7.
[7] B. A. Thomas. New York Med. Jour., 1907, ii, 688.
[8] Phila. Med. Jour., 1901, ii, 193.
[9] Annals of Surgery, 1904, i, 7.

The lesions found may be seen in the annexed table:

Lesion.	Cases.	Recovered.	Died.	Mortality. Per cent.
Cholecystitis alone	4	2	2	50.00
Cholecystitis and empyema of gall-bladder	3	0	3	100.00
Cholecystitis, empyema, and peritonitis	4	2	2	50.00
Perforation with peritonitis	6	4	2	33.30
Perforation (found only at autopsy)	4	0	4	100.00
Total	21	8	13	61.90

The bacteria found were as follows:

Bacterium.	Cases.	Recovered.	Died.	Mortality. Per cent.
Typhoid bacillus (pure culture)	9	5	4	44.40
Typhoid bacillus and colon bacillus	1	0	1	100.00
Paracolon bacillus	1	0	1	100.00
Not recorded	10	3	7	.....

In two of the patients treated by cholecystectomy two small calculi were found in each gall-bladder. In the case of the patients tabulated as having been treated by cholelithotomy there were 152 and 150 gallstones, respectively. Thus, in only 4 cases (19 per cent. of the total number) were stones found in the gall-bladder. Among the 216 cases of infection of the biliary tract studied by Kelly,[10] 84.2 per cent. had calculi; this would make it appear that calculi do not particularly predispose to gall-bladder complications during typhoid fever. That the typhoid bacillus, however, is a rather frequent factor in the causation of subsequent inflammatory affections of the gall-bladder is proved by Dr. Kelly's[11] earlier investigations, which showed that this organism was present in 10 per cent. of the cases studied bacteriologically.

The symptoms are recorded in all but 2 patients. There are two quite distinct classes of cases: In the first there has been a more or less gradual onset of abdominal pain, fairly well localized (by patients who were conscious) to the right hypochondrium, accompanied by localized tenderness, and frequently by a palpable mass, easily recognized as the distended gall-bladder. If operation has been undertaken at this stage, there has been found (*a*) cholecystitis; (*b*) empyema; or (*c*) empyema and commencing suppurative pericholecystitis. In the second class of cases the symptoms already mentioned have existed for a period varying from a few hours to a week or ten days, when suddenly there has been an acute attack of abdominal pain, accompanied by a fall of temperature (noted in 8 out of 10 cases), and sometimes by sweating; these signs being gradually followed, when immediate operation was not undertaken, by a secondary rise of temperature, a spread of the pain and tenderness over the whole abdomen, and an increasing distention of the intestines. In one patient (Alexieff) a gall-bladder, which had been

[10] Amer. Jour. Med. Sci., 1906, cxxxii, 755

[11] Ibid., 447

palpable for some days, suddenly collapsed and could no longer be detected on the occurrence of perforation. In a few cases, as in the first patient now reported (Davis), the delirious or semicomatose condition obscured the symptoms usually preceding perforation, and the only physical signs detected resembled so closely those of intestinal perforation that the diagnosis naturally inclined to this much more frequent complication.

It is interesting to note that out of the 21 cases reported, a correct diagnosis was made in 11; in 9 cases the diagnosis made was intestinal perforation; in 1 case (a woman eight months pregnant) the only diagnosis was peritonitis.

As to the frequency with which the gall-bladder is involved in typhoid fever, no satisfactory statistics have been published. At the Episcopal Hospital the reports do not mention the complication of cholecystitis prior to the year 1905. From January 1, 1905, to October 1, 1907, there have been treated in our wards 2864 patients with typhoid fever. Among this number there were 243 deaths (a mortality of 8.48 per cent.). There are recorded 18 cases complicated by cholecystitis (about 0.62 per cent. of the whole number of cases); and among the cases thus complicated by cholecystitis there were 4 deaths. This, however, by no means implies that the deaths were due to the cholecystitis, since patients who at one stage presented symptoms of cholecystitis might recover from those symptoms and die at a later period from the toxemia of typhoid or from asthenia; yet they would still be reported as cases complicated by cholecystitis. Only two of these cases were considered serious enough for the thought of operation to be entertained; and it appears a mere coincidence that these two should both have been observed during the present year, within a few weeks of each other.

CASES OF TYPHOID FEVER AT THE EPISCOPAL HOSPITAL, PHILADELPHIA.

Year.	Cases.	Deaths.	Mortality. Per Cent.	With gall-bladder complications. Cases.	Deaths.
1905	981	85	8.66	8	1[12]
1906	1339	107	7.99	6	2[13]
1907 (to Oct. 1)	544	51	9.37	4	1[14]
Total	2864	243	8.48	18	4

[12] Recovered from cholecystitis before death. No operation.

[13] Neither patient was operated on. Autopsy only in the second case; this showed no peritonitis, and that death was due to pneumonia.

[14] Operation by Dr. G. G. Davis. Perforation of gall-bladder found postmortem.

TWENTY-ONE OPERATIONS ON THE GALL-BLADDER DURING TYPHOID FEVER.

Sex.	Recovered.	Died.	Total.	Mortality. Per cent.
Male	3	8	11	72.7
Female	5	5	10	50

Age.	Recovered.	Died.	Total.	Mortality. Per cent.
Less than 10 years	1	0	1	00.0
10–19 years	1	4	5	80.0
20–29 years	2	3	5	60.0
30–39 years	2	4	6	66.6
40–46 years	2	2	4	50.0

Stage of disease.	Recovered.	Died.	Total.	Mortality. Per cent.
First week	0	1	1	100.0
Second week	0	3	3	100.0
Third week	1	4	5	80.0
Fourth week	1	1	2	50.0
Fifth week	2	3	5	60.0
Sixth week	3	0	3	00.0
Ninth week (relapse)	0	1	1	100.0

(Stage of disease not recorded in one patient who recovered.)

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THE DIAGNOSIS AND TREATMENT OF SYPHILITIC LESIONS OF THE NERVOUS SYSTEM.[1]

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It is of the utmost importance to make the diagnosis of syphilitic lesions of the nervous system as early as possible, for it is in the primary stages that therapeutic measures are of most benefit. That the diagnosis of such lesions presents many difficulties is evident, since there is no part of the nervous system that may not be attacked. Certain symptoms are present, however, in nearly all cases, and it is principally by the recognition of these that we are able to make a proper diagnosis. The better to understand what symptoms are possible in syphilis, an effort will be made to trace the course and nature of the disease and the method of involvement of the nervous system.

In only a few of the large number of persons infected does the nervous system become secondarily diseased. The important question, then, is, Why should one person have his nervous system affected and not the other, and what influence, if any, does treatment have upon the prevention of such involvement?

Repeated instances have been recorded in which a number of persons not at all related have become infected from the same source and subsequently developed general syphilis and, later, specific disease of the nervous system. Here there are two possibilities: one, the most probable, that the specific infection was of such character that it had a special affinity for these structures; and second, that there was lessened resistance of the nervous system to the infection. It has always been one of the old theories that only certain forms of specific intoxication will cause disease of the nervous system; the best proofs of this have been such instances as quoted above. That such is the case there can be no doubt, but such a theory will hardly answer for all cases. There is no question that specific manifestations at the present time are not as severe as they have been. This is due partially to the better recognition and earlier and better treatment of the disease, and also to the fact that all diseases have a tendency to lessen in intensity in the course of time.

It is probable that the most important factor which determines whether the infected person will have future involvement of the nervous system is the lessened resistance of these structures, or

[1] Read by invitation at a meeting of the Lackawanna County Medical Society, Scranton, Pennsylvania, October 22, 1907.

possibly a special predisposition. Just what determines this is, of course, difficult to tell, but it must be that certain parts of the anatomy are less resistant than others. Hereditary and family influences contribute to this to a large degree. Erb records instances of specific infections in families in which all had tabes dorsalis following infection from the same source.

It is of importance to know in what percentage of cases of syphilis the nervous system becomes diseased. This can never be determined with accuracy, and we must rely principally upon the statistics to be obtained from institutions. Erb, for intsance, collected some years ago a history of many cases of specific infection, and of these, in about 1 to 5 per cent. tabes followed. In how many, however, there were other involvements of the nervous system is not recorded, but judging from the fact that tabes is no more frequent than other cerebrospinal diseases, it is probable that a fair percentage would be about from 5 to 10 cases in 100. This, of course, is only conjecture.

It is also a question whether or not early treatment of the disease will prevent such future involvement. That it does so in the majority of cases there is no doubt, but in many instances it is of no avail. In one case, a physician who was infected in his finger, the most active treatment was immediately instituted and faithfully kept up for three years. While still taking medicine, three years after the infection, the patient suddenly had diplopia, which was followed by other basal symptoms, this terminating, in two years, in multiple cerebrospinal symptoms and finally in death. In another case, also a physician, who was similarly infected, the symptoms of basal involvement appeared a year after the infection, while he was undergoing the most vigorous treatment. The disease is still progressing, and antispecific treatment seems of no avail. These two acute cases are quoted, among others which have occurred in my experience, to show the uselessness of treatment in certain forms of syphilis.

It is, however, in the more chronic cases, or in those in which the symptoms of cerebrospinal involvement appear many years after the infection, that this question arises. In many, antispecific treatment had been instituted and carried out vigorously, and the patient pronounced well, only to have some part of the nervous system become diseased years afterward. The conclusion is forced upon us that in some forms of syphilis the cerebrospinal system will become diseased whether or not treatment is instituted, and no matter how vigorously.

After an infection the nervous system may be involved almost immediately, in the midst of treatment, or may not for some years, sometimes as many as twenty. It is generally supposed that the earlier the cerebrospinal symptoms appear the more severe the infection. Considering, however, that the symptoms produced

years after the infection are about of the same severity and character as they are in the early periods, there is probably little difference.

Pathologically, in the early forms, no matter where the nervous system is involved, there is always a round-cell infiltration about the bloodvessels and in the pia, this being especially true of the base of the brain and the posterior part of the spinal cord; and there is also a secondary endarteritis and permanent thickening of the meninges. Rarely, small gummas will be found, but these are not as frequent in the nervous system as is commonly supposed. More rarely, there is also an involvement of the substance of the cord or brain. When such is the case, in the former there is very often a total inflammation, this resulting in myelitis; if in the brain, the cortex and subcortex are diseased, this causing the symptoms of diffuse encephalitis. It is characteristic of these acute inflammations, such as myelitis and encephalitis, that the symptoms appear suddenly.

When this disease comes on many years after the infection the usual pathological findings consist in a sclerosis of the vessels and thickening of the meninges, and sometimes in gummas, but it is principally to the results of the endarteritis that the symptoms are due. There are, however, other forms of disease resulting from specific infection which are caused not by inflammation, but by gradual changes in the nervous structures, thought to be the result of toxins. These changes mostly occur in structures which have more or less functional relation, and are called systemic, the best example of this being locomotor ataxia or tabes dorsalis.

It can be readily understood, then, that, so far as the symptoms of specific disease of the nervous system are concerned, the majority of cases will show an involvement both of the brain and of the spinal cord, and that the focal symptoms will depend upon what particular portions of these structures are most diseased. No matter what the extent of the affection, there is in every case an infiltration of the meninges, and this in itself will nearly always give certain symptoms. There is a tendency, however, for certain forms of involvement in those cases in which the symptoms manifest themselves within a few years after the infection, and these will be discussed first.

Among the earliest is a myelitis, this usually coming on suddenly and in most instances affecting the lower thoracic or thoracic-lumbar region, and often giving the symptoms of a complete transverse myelitis, there being loss of power in both lower limbs, of the bladder and rectal functions, and of sensation. In the majority of cases some return of power can be hoped for, especially if vigorous treatment is promptly instituted. Added to the symptoms of the myelitis there will be those of meningeal involvement, such as girdle sense and pain.

Other common early manifestations, sometimes appearing in the midst of treatment, are those the result of multiple lesions of the brain and cord, giving diffuse symptoms depending upon the location of the lesions. There will nearly always be motor and sensory involvement of irregular character, with the addition of meningeal symptoms of the base of the brain and of the cord.

Hemiplegia is also common in early syphilis, this resulting from early endarteritis. In fact, hemiplegia occurring in young adults below forty years of age, is nearly always syphilitic in origin, but the most common manifestations of syphilis are those the result of meningeal involvement of the base of the brain or of the spinal cord.

Meningitis of the base of the brain is nearly always greatest around the chiasm, and it is because of this that the optic, oculomotor, and sixth nerves are commonly diseased in syphilis. Whether it is because of this involvement of the optic nerves, or because the specific toxin has a special predilection for the iritic muscles, disturbances in the reactions of the pupils with irregularities in their margins are the most common and constant symptoms of syphilis. Such general diseases as syphilis nearly always affect those structures which are most commonly used, and it is, perhaps, because of this that the iritic muscles are so early diseased. In most instances there will be found irregularities in the margins, with a slow reaction of the pupils both to light and to movement of the eyeballs, and sometimes, in the later stages, a failure of response of the pupils to light, with preservation to movement of the eyeballs, or the Argyll-Robertson pupil. There is no disease which will so constantly give these symptoms, and there are no symptoms which are so constant or which can be so much depended upon.

Oculomotor palsy, unilateral in type, occurs most frequently in syphilis. In fact, it has been termed its *sign manual*. Of the cranial nerves, however, the sixth nerve is probably the most frequently diseased, it causing double vision. This, as a rule, lasts only a very short time, appearing and reappearing, until finally it will be permanent. Paralysis of any of the other cranial nerves is a rare manifestation. Meningeal involvement in the spinal cord is nearly always greatest in its posterior portion. Because of this, girdle sense and pains referred to various parts of the limbs are common symptoms.

Of the diseases which have a tendency to become manifest many years after the infection, that is, after the fifth year, while there is in every case an affection both of the brain and the spinal cord, there is nearly always a preponderance of the disease in one of these structures. When the brain is mostly involved there will be, besides the irregularity of the pupils and the slowness of reactions to light and movement, other symptoms depending upon the location of the lesion. These may consist of a gummatous deposit in most any portion of the brain or of a basal infiltration, besides those

diseases resulting from the early endarteritis. For instance, a man, aged thirty-two years, began to have headache, nausea, some dizziness, with disturbance of the sense of position, of movement and localization in the right upper limb, and inability to recognize objects placed in this hand. A tumor, probably a gumma, was present in the left parietal cortex. Mercurial inunctions caused total disappearance of the symptoms, with some remaining pupillary irregularity.

An example of cortical meningeal involvement, with the addition of gummatous deposits in the brain tissue, is that of a man, aged forty-five years, already reported, in which there was present the symptoms of a lesion in both the parietal and the motor area. The patient had Jacksonian convulsions in one upper limb, accompanied by the most excruciating pain, which was followed by a loss of power and increased reflexes, besides the usual symptoms of brain tumor. This man was operated upon twice and considerable gummatous tissue removed. At necropsy an extensive meningitis was found over both sides of the cortex, the meninges in places measuring almost one-half inch in thickness. Besides, the brain was infiltrated with gummatous deposits and the skull was carious almost in its whole thickness.

An example of basal involvement, this probably affecting the meninges and also the pons, is that of a physician, a man aged forty years, who, two years after infection, became gradually stupid and dull and had to abandon his practice. He then developed difficulty in talking, eating, and swallowing, and weakness in the right half of the body, besides paralysis of the fifth and sixth nerves.

Syphilis may sometimes cause the general symptoms of brain tumor without any special localizing phenomena. Such, for instance, is a man, aged forty years, who developed, some years after specific infection, headache, nausea, vomiting, and bilateral choked disk. Besides, the pupils were irregular and the reactions slow. Antispecific treatment in this case has proved of the greatest benefit. In another case, studied in the Philadelphia Hospital, and subsequently reported by Dr. Mills, a young man had all the symptoms of a cerebellar tumor, only to find at necropsy multiple gummatous deposits throughout the brain.

It is characteristic of brain symptoms, just as it is of those the result of basal lesions, that they may appear for a time and then disappear, only to reappear again and remain more or less permanently. Such was the case of a young woman who, when seen, presented all the symptoms of acute confusional insanity. Physical examination, however, disclosed irregular pupils, with slow reaction to light, and the history, as obtained from one of the parents, of temporary diplopia, associated with motor aphasia and some difficulty in writing. Both the aphasic symptoms and the difficulty in writing remained only for a short time and then disap-

peared. A diagnosis was made of a specific disease, this being based upon the pupillary condition and temporary appearance of symptoms. Later, specific disease was acknowledged.

The above cases are quoted, among others, as examples of the different lesions that may be expected in syphilitic disease of the brain. The characteristics of all were a more or less gradual appearance, and, besides the definite symptoms due to focal involvement, the presence of the pupillary irregularities and disturbance in their reactions.

In those instances in which the spinal cord is mostly involved there may be very diffuse symptoms, but in nearly all there is some meningitis, this causing girdle sense and pains. That the symptoms, however, may be diffuse, is exemplified in the following case:

A man, aged forty-three years, began with weakness in the right leg, which was followed by pain in the right buttock and along the course of the right sciatic nerve, accompanied by a feeling of numbness in the whole right lower limb and a tight feeling across the knee. The patient's attitude and gait were such as are present in sciatica. He also had irregular pupils with slow reactions, and a history of temporary diplopia. The case was variously diagnosticated at different times, and both a spinal cord tumor and hip-joint disease were suspected, but after having been placed on mercurial inunctions, he became entirely well.

Another case is that of a man, aged thirty-one years, who had rheumatic pains in the left thigh and left lower limb, with pain along the sciatic nerve. This limb was weak and somewhat atrophic and the tendon reflexes were absent. He had, besides, the usual specific pupillary phenomena. Under mercurial inunctions the symptoms disappeared almost entirely.

In the following case the symptoms were almost totally confined to the bladder functions: A man, aged about forty years, six years before observation, began to find it necessary to urinate oftener than normal, this frequency steadily increasing. When examined there were no other symptoms present besides the irregularity of the pupils, with slow reactions to light and to movement. Under antispecific treatment the bladder symptoms are steadily disappearing.

Disturbances in bladder and rectal functions, especially the former, are very common in spinal specific infections, but sometimes, as in the case quoted above, they may be the only symptoms indicating such disease.

The above three cases are quoted as being typical of spinal specific disease. In all there was a preponderance of meningeal symptoms, with disturbance of either bladder or rectal functions, with the addition of pupillary symptoms, and it was upon these that the diagnosis of syphilis was made.

Cases are observed in which there seems to be an equal involvement of the brain and the spinal cord, but even here one of these

structures may be more diseased. The symptoms will, as a rule, be multiple, and there will be, besides the specific pupillary phenomena, disturbance of mentality, irregular motor and sensory symptoms, and involvement of the bladder and rectum.

An example of such a case is that of a farmer, aged forty-two years, who began with weakness and numbness in the lower limbs, with increased urination and constipation, loss of memory, and changes in disposition. Examination showed, besides motor and sensory symptoms, irregularities of the pupils. Antispecific treatment in this case was not of much value.

So far, only those cerebrospinal syphilitic diseases have been discussed which are the result of the direct pathological processes produced by such infection, while other diseases, as general paresis, tabes dorsalis, and posterolateral sclerosis, are supposedly produced by its toxins. Their symptoms, as a rule, come on many years after the infection, at least so far as their appreciation is concerned. It is curious that a person may be infected with syphilis and have no symptoms for a number of years, and then may develop posterior sclerosis. This does not seem logical, and it is probable that if our methods of observation were better we should be able to demonstrate some symptoms, for the syphilitic toxin does cause early and constant changes.

That such is the case has been demonstrated in more recent years by the examination of the cerebrospinal fluid. It has been found that in such diseases as tabes dorsalis and general paresis, in which it is supposed that the cause is specific, there is always an increase in the lymphocytic elements. Indeed, recently a certain form of Spirochæta pallida, which is supposed to be the cause of syphilis, has been demonstrated in the cerebrospinal fluid. This increase in the lymphocytic constituents occurs in other diseases, and as yet cannot be absolutely depended upon. At a recent meeting of the Philadelphia Pathological Society, Dr. Simon Flexner demonstrated a rapid method of obtaining Spirochætæ pallida from primary and secondary manifestations. At necropsy this organism has been found in gummatous deposits of the brain and cord, and it is probable that, if carefully looked for, it will be commonly found. So far as an aid to the clinical diagnosis of this disease, examination of the cerebrospinal fluid, while of great, is not as yet of positive, value.

TREATMENT. It is in the early stages of this disease, in the period of round-cell infiltration, that antispecific treatment is of the greatest benefit, and treatment should be promptly and vigorously instituted. The best results can be obtained from inunctions of mercury, a dram a day, to be rubbed in in different places. The best plan to follow is to apply this daily, with the exception of Sunday, and to keep it up for a period of about six weeks; if any improvement can be hoped for, it will be manifest in that time. If,

at the end of this period, there is no improvement, such treatment might as well cease.

It is always a good precaution to have the patient's teeth carefully looked after before treatment is instituted. During the treatment a borax mouth wash should be used, and the patient not allowed any dietary indiscretions, although special dieting is not necessary. It is also advisable to take the patient away from all work, hospital treatment being the best. A hot pack, given about two or three times a week, will aid in the absorption of the mercury.

It has been my experience that whenever any benefit has been obtained, it has been the result of the use of mercury, and not of iodides. Even in the specific manifestations which come on many years after the infection, mercury is the only drug which will be of benefit. Iodides probably do good, and should be given either in conjunction with the mercury or, better still, after the mercurial treatment. It is a good plan to give them in increasing doses, 50 to 70 grains a day being quite enough. The habit of giving large doses of iodides is quite prevalent in this country, while abroad smaller doses are the rule.

THE NEURASTHENIA OF AUTO-INTOXICATION.

By Thomas J. Orbison, M.D.,
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The symptom-complex of neurasthenia may be likened to a fairly well-defined body of water fed by many underground channels that in their turn flow from hidden sources. One of the largest of these tributaries is heredity, which is fairly well charted. Besides this there are, doubtless, many others. Of these I believe a very important one, indeed, has its source in gastro-intestinal auto-intoxication.

There are two ways by which man may be poisoned—either by a poison from without or by a failure to get rid of the poisons that are formed inside him, provided it is possible for the body to take them up again.

That neurasthenia may be caused by the former has been accepted. Can it be induced by the latter? Before answering this it will be necessary to answer the following questions: Does the body form poisons within itself? If so, where may they be found? May they be taken up again into the body owing to a vicious condition of the bodily hygiene or a pathological condition of certain secreting surfaces? If they may be so taken up, will they give rise to the symptom-complex of neurasthenia?

On the one hand Oppenheim[1] distinctly and explicitly stands for the negative. He says: "The opinion that this disease (neurasthenia) is due to auto-intoxication resulting from disease of the gastro-intestinal tract does not seem to me to be sufficiently well established." He does not even refer to the urinary findings and their relation, in neurasthenia, to intestinal putrefaction

There is a middle ground to this subject, where many teachers are found. Osler[2] does not mention auto-intoxication as a cause of neurasthenia. Like Oppenheim he seems to look upon neurasthenia as a disease of the mentality caused by worry, overwork, and the like; or else the neurasthenic condition is produced by certain poisons from the outside—such as morphine, cocaine, and alcohol. He refers to gastro-intestinal neurasthenia as nervous dyspepsia and says it is due to worry or nerve tire. No mention is made of the urinary findings in the disease or the fact that they may point the way to one of its causes. Musser[3] does not mention any connection between neurasthenia and auto-intoxication, nor does he deny it.

Bouchard[4] as distinctly affirms as Oppenheim denies that auto-intoxication is an important cause of neurasthenia. So much for the academic side of the question.

Does the body form poisons within itself?

Referring to indicanuria, Musser quotes von Jaksch who states that large quantities of indican in the urine imply that abundant albuminous putrefaction or putrid suppuration is in progress in the system. Musser adds that it must not be forgotten that indicanuria will often arise in simple constipation. Osler tells what indicanuria is, states the origin of the indican and says it is a product of the decomposition of albumen under the influence of bacteria. He leaves the subject there.

Boas[5] quotes Jaffe, Salkowski, Bauman, Brieger, and Senator to prove that indican in the urine is derived from the intestines and is a normal product, but becomes pathological in excess. It is normal from 5 to 20 mg. daily; pathologically, it may reach 100 to 150 mg. Nothnagel[6] quotes Jaffe, who observed that obstruction of the small intestine causes marked increase of indican. Constriction of the large bowel does not cause so much increase. Indican is increased in diarrhœa as well as in constriction and constipation due to bowel stasis, provided the small intestine is at fault, that is, in typhoid when there is disorder of the mucosa, and in other diseases confined or localized in the small intestines. Ortweiler found that increased peristalsis alone does not necessarily cause increased indican in the urine, and may cause its absence owing to the sweeping away of the putrefactive material in the intestines.

From all of this it seems certain that the body does form poisons

[1] Diseases of the Nervous System.
[2] Practice of Medicine.
[3] Medical Diagnosis.
[4] Auto-intoxication in Disease.
[5] Diseases of the Intestines.
[6] Diseases of the Intestines.

within itself. What are these poisons and may they be taken up by the body instead of being expelled as they should be?

Bouchard has isolated seven distinct poisons in the urine alone, and shows their action on living tissues. He proves the toxicity of the blood itself in its capacity of carrier of the poisons to the various excretory organs. He convicts the bile of a tremendous toxicity. Graphically, he gives the relative toxicity of urine and bile and shows mathematically that "if all the bile secreted by the liver passed directly into the blood of a man he would be poisoned by his own bile in less than nine hours. If all the urine that his kidneys secreted passed directly into the blood he would be poisoned by his own urine in two days and six hours." He goes minutely and exhaustively into the subject of auto-intoxication, and leaves not a doubt of it in the reader's mind. Müller and Ortweiler consider it certain that the absorptive faculty of the bowel plays the prominent part in the transfer of poison to tissue.

It seems, then, that the body does form poisons within itself, that they may be found in the urine, bile, venous blood (especially just before it gets to the liver), and in the arterial circulation itself; likewise, that these poisons have been taken up from the gastro-intestinal tract and are constantly in the process of being excreted by the various emunctories. In the healthy man compensation is balanced; the out-go equals the amount formed. But, if any one or more pathological conditions exist—a failure to throw off the various poisons in adequate amounts, loss of the integrity of some mucous surface, stasis in the small bowel from any cause, an excessive intake of vicious material—what then occurs? The intestinal tract is an ideal laboratory for the growth of bacteria. It is a flexible tube, accessible at both ends, kept at a certain temperature and filled with the materials suitable for putrefaction. A large section of it is lined with a secreting surface. It does not take much imagination to picture the condition of the man who is absorbing more of these various poisons than be can throw off. The symptoms would naturally vary in degree and severity according to the individual's personal equation, the excess of poison, and the susceptibility of his tissues.

While it would be ridiculous to assert that every neurasthenic is such because of absorption of toxic material, in other words that auto-intoxication is its only cause—yet it does not seem ridiculous to claim that very many neurasthenics are such wholly or in part owing to auto-intoxication, when we realize the chances at hand for inducing this condition and consider what the results of it would naturally be. This hypothesis is confirmed and strengthened by clinical experience. In this connection I wish to cite an instructive case:

Case I.—C. P., was twenty-nine years old when he came to me. His occupation is sedentary, although he gets plenty of out-of-door

exercise. His family history shows a distinct mental taint on the maternal side. Otherwise the history is good.

He had pleurisy, scarlet fever, and typhoid fever fourteen, eleven, and eight years ago, respectively. Coming, as they did, in the time of his adolescence they may have had a good deal to do with his case as predisposing factors. His general health, other than these, has been good up to about two years ago. There is no history of venereal disease or sexual excess. He has enjoyed plenty of out-of-door exercise—especially rowing and walking.

For about two years he has complained of not feeling well. When asked to be more explicit he said he felt sleepy most of the time, complained of a lack of tone and inertia associated with mental heaviness, constant fagged-out feelings, fulness in the head, and a generally tired-out feeling. He cannot exert himself or do things with any degree of enthusiasm as he would like to do and used to do. He has headaches and neuralgias. He becomes very much depressed at times and does not feel the normal mental exhilaration and joy of life he was accustomed to feel. He has no cause for worry and his work is not exhausting. His bowels are generally constipated. He says he has noticed a lump in the right abdominal region that varies in size from time to time. He has gone the rounds of physicians and has been treated accordingly.

His condition has been diagnosticated neurasthenia, hysteria, general nervousness, and hypochondriasis by competent observers of repute. He says that if his present condition should continue much longer he would feel that life would be too much of a burden.

His muscular condition is excellent—no flabbiness, no pendulous abdomen. The heart and lungs are normal. There is no abnormal position of any of his organs. The tendon reflexes are active but not exaggerated. He exhibits a pretty constant series of habbit spasms, especially of the orbicularis palpebrarum and facial muscles. These express themselves at times as grimaces and are bilateral. There seems to be a desire to keep certain of the eye muscles on the stretch. He has had a number of attacks of sciatica and as a result there is some spinal curving. Peristalsis is sluggish as demonstrated by the phonendescope. Rectal examination shows some dryness of the contents and a good deal of adhesion of fecal material to the bowel walls. The sphincter muscles are exceedingly active and over-tense. The eyes were examined by Dr. J. T. Carpenter who reported that the examination was negative as far as it showed no adequate cause for his nervous condition.

The examination of the urine was negative as far as the ordinary clinical technique goes and showed no abnormality.

Here was a case of, so-called functional, neurasthenia; it had the symptom-complex without adequate anatomical findings to account for them, apparently.

The treatment was directed at the condition of constipation. The

result was good, as far as it went; some increase in weight, a better hygienic condition of the bowels and kidneys. It was not satisfactory, however, as no definite demonstrable cause for his trouble had been found.

A more extended examination of his urine was made and at once pointed to the real cause. There was a report from the Philadelphia Clinical Laboratory of "very high quantity of indican for normal twenty-four-hour quantity of urine." He was passing about fifty ounces daily. Thus, it was pointed out, there were two large causal factors in the case to be considered—heredity and auto-intoxication.

Another physical examination was made to find, if possible, any anatomical basis for bowel stasis or obstruction. The abdominal lump was recalled and the patient directed to return at its next reappearance. This he did and a movable, balloon-like tumor was marked out in the region of the appendix. It was partially collapsible, but not entirely removable by palpation. Two high enemas were injected to clear out the lower bowel and aid the examination. The notes read "the water ran in easily traversing the descending, transverse, and ascending colon in turn arriving at the cecum. After the water was evacuated examination showed an irreducible mass in the region of the appendix. The bowel was flattened and the mass distinctly felt rolling under the fingers." Subsequently, four examinations of the urine exhibited indican in excess.

The facts were these. There was a constriction in the neighborhood of the appendix resulting in bowel stasis. This was considered sufficient to set up intestinal putrefaction with absorption of the resulting poisons; these, in turn, were held responsible for his neurasthenic condition.

Dr. John Gibbon was consulted. His examination fully upheld the diagnosis and his opinion was that the nature of the obstruction was the presence of adhesions in the appendicular region. We held to this although there was no history of appendicitis or acute pain in that part of the abdomen. A second examination was made a month later and the same condition found.

Operation was advised and carried out. The notes of it include the following: "The bowel and appendix were exposed. The latter was long, twisted, and bound to the former by adhesions. The bowel was much constricted by these adhesions. Nothing else was found to be abnormal; the kidney and gall-bladder were in a good position. Adhesions were broken up and the appendix removed. The tip of it was quite injected with hemorrhagic spots, but otherwise normal. In addition to the operation the sphincter ani muscle was thoroughly stretched. Just prior to the operation a differential blood count was made in the Pennsylvania Hospital laboratory; this was normal. Nine days after the operation the urinary indican was about normal. One year later the report is: "Examination of the urine shows no excess of indican." The

patient is feeling like a new man and has recovered entirely his joy of living, enthusiasm, and right feeling—and he has not changed his occupation or mode of life, except that his bodily hygiene is kept as it should be.

This is the record of a case of neurasthenia due to auto-intoxication. It had persisted for years until the cause of the auto-intoxication was removed and then cleared up.

I have appended eight cases (including the one herein enlarged upon) of neurasthenia occurring in private practice in Philadelphia, in which the symptoms pointed to auto-intoxication and the diagnosis was corroborated by the urinary findings. Of these two have been cured and are entirely free from symptoms. One may be called very much improved—in this case there have been no symptoms for some months, but this is hardly long enough to enable one to claim a permanent cure as yet. Three have been improved by a course of treatment directed against the condition of bad bodily hygiene. Two have not been under treatment long enough to make any claims as to results. There is great satisfaction in finding a tangible cause for these cases that are without anatomical findings—a sign post that may point in the direction of health to many otherwise chronic cases.

One point in addition to this question is pertinent: Why is it that, when the urinary findings begin to indicate that the poisons of putrefaction are no longer excessive, the neurasthenic condition continues, in many cases, for a long time? I wish only to touch on one point relative to this—on the withdrawal of a poison that has been absorbed for a long time, the nerves seem to cry out in distress. This is a common experience and may have some bearing on these cases.

CASE II.—M. J. O'M. Male, aged thirty-five years, occupation architect and builder. Generally very healthy. He has been a regular but never an excessive drinker of alcoholics, an excessive smoker for years, and inclined to worry over trifles even though his business has prospered.

In February 1906, following a spell of continuous hard work, he began to feel tired, irritable, and unable to do his work satisfactorily. This was associated with a nervous dyspepsia and backache in the lower dorsal region. He was possessed by terrors of various kinds, especially if left to himself. His heart would palpitate easily and that would increase his fear.

His weight is 174½ pounds. General physical condition is good. His abdomen is pendulous, and there is a slight lateral curvature of his vertebral column. All his reflexes are exaggerated and jerky.

Report of urinary examination by the Philadelphia Clinical Laboratory: "Indican is high for normal twenty-four-hour amount of urine; otherwise normal."

Treatment consisted in correcting the spinal asymmetry and over-

coming the sluggish tendency of the bowels by massage, medication, rest, and change of scene. He has become entirely well.

Case III.—Mrs. C., aged forty-three years. Father died of nephritis and cirrhosis of the liver. Her mother has diabetes. Pneumonia at ten years of age; diphtheria twice; whooping-cough and measles during early childhood.

As a young girl she was very active and easily overwrought. When about eighteen years old she had nervous prostration. Since then she has had attacks of nervous dyspepsia. She has grown quite stout in late years. During the last six months she has had persistent constipation, mental depression, intense lassitude, and headaches; gets tired upon slight exertion. With this there has been a harrassing fear of being alone or in the country and a feeling that she must be constantly on the go even though felling tired out.

Weight 185 pounds (30 pounds too much). Abdomen pendulous and abdominal muscles flaccid and atonic. Conjunctivæ are congested and icteric; tongue coated. Reflexes are very jerky.

Urinary examination by Philadelphia Clinical Laboratory, November 8, 1906, showed: "Total amount of twenty-four-hour urine 500 c.c.; marked increase in indican; slight increase in phenol; total sulphates equal 1.33 grams; conjugate sulphates equal 0.177 gram; preformed sulphates equal 1.153 gram; ratio of preformed to conjugate equal 6.5 to 1; normal ratio is 10 to 1."

Although there was no *actual* increase here in the amount of sulphates excreted there was a relative increase in the conjugate over the preformed. This goes hand in hand with increase in indican and phenol and helps to confirm the diagnosis of intestinal putrefaction.

Treatment consisted in correcting the intestinal sluggishness, by intestinal antisepsis, appropriate diet and exercises, together with massage.

The report of urinary examination, January 15, 1907, was normal except a slight deficiency in amount; she had increased the amount from 562 to 900 c.c. Her general condition was better in every respect. Her weight fell from 185 pounds to 174¾ pounds. There is yet a good deal to be done before she feels normal—habits to overcome, etc.

Case IV.—Mrs. B., aged twenty-eight years. Father died of fatty degeneration of the heart. Her mother is of a distinctly hysterical make up and always has been. Even when Mrs. B. was a child her mother made no attempt to control her outbursts. She was born at seven months. Has always been constipated. As a child she craved "sweets" and was allowed to eat them at will. She has never been robust though she has never been severely ill. She was married at twenty-one years of age and become pregnant three years later; during her pregnancy she felt better in every respect.

From childhood she has suffered from apprehensions. Has always been very impressionable and in later years this has led

toward hypochondria. At times she has the feeling of a tight-band about the head; she becomes very depressed and weeps easily. There have never been true delusions or hallucinations, although about six years ago she began to be suspicious. This passed away. She is very thin. Her abdomen is pendulous. The left kidney is much too low. Heart and lungs are normal. Reflexes are generally somewhat increased. There are no hysterical stigmata other than the "fillet."

Examination of urine shows a trace of albumin and indican in excess; otherwise normal.

CASE V.—Mrs. B., aged forty-one years. Mother died of gastro-hepatic intestinal disease. Father died of a complication of grip, erysipelas, and senile dementia.

Was married at twenty-two years of age; before this she was always healthy. Since the birth of her first child she has never been well. She had five pregnancies in eight years. About one and one-half years after the birth of her last child she had purulent salpingitis and was operated upon; both tubes and ovaries were removed. She was in bed eighteen months. At this time she was dropsical. It was several months later when she began to walk about. Shortly after she began to walk she developed hystero-epilepsy. During some of the attacks she would fall and has broken various bones. These lasted for nine years after which their character changed—at times she has intense pseudo-angina following the attack, at other times she comes out of them quietly. About this time she developed marked dyspeptic symptoms with constipation. For the last three years she has had constipation and diarrhœa in turn. When constipated there is intense headache with nausea and vomiting associated with scanty urine. After the diarrhœa there is free micturition and relief from headache. She complains of mental depression, inability to attend to her household duties, and intense headaches. There is considerable ballooning of the bowels at times.

Examination of urine showed indician "high for twenty-four-hour amount."

Treatment was directed against the intestinal putrefaction with the result that she no longer has the epileptoid attacks, is almost entirely free from her headaches, and is able to attend to her duties. I showed this woman to Dr. Wm. G. Spiller, who concurred in the diagnosis of hystero-psychic-epilepsy with intestinal auto-intoxication as its basis.

CASE VI.—Mrs. C., aged thirty-eight years. Good as to health; both parents were emotional. She was perfectly well up to fifteen years of age when she had a "nervous breakdown." This was characterized by unreasoning fears of various kinds. There were no hallucinations or delusions. She had fits of depression and crying. This lasted about one year and there were hysterical stigmata exhibited, that is, spells of apparent unconsciousness that were not

epileptic. She was married at eighteen years of age; has never been robust since. At times she will feel quite well and then have a period of nervousness and mental depression. She complains of lassitude, tired feelings, mental depression, and fear—fear of crowds and being alone. Her mode of life does not account for them—there is no apparent cause. Her symptoms have been more severe for the last two years. Her bowels have been very irregular up to two months ago; since then they have been regulated by internal treatment. Sleep is very unsatisfactory and appetite poor.

General physical condition is good. Eyes are watery. Conjunctivæ are hypesthetic. Reflexes are exaggerated. Heart and lungs were normal.

Examination of urine at the Philadelphia Clinical Laboratory shows a trace of albumin and indican high for twenty-four-hour amount of urine. Otherwise it was normal.

Treatment was directed against the intestinal putrefaction and urinary insufficiency, and the result has been very satisfactory as far as it has gone—the length of time has not been sufficient to prove permanent good.

Case VII.—Mrs. S., aged thirty-four years. Mother died of cancer. Father alive. One sister died of tuberculosis. One sister and three brothers are alive and healthy. From childhood she has been of a very neurotic disposition. Up to her first menstrual period she had what she calls "spasms;" during them she was unconscious. These lasted from her ninth to her fourteenth years. For the last nine years she had fits of mental depression. About one year ago she had a hysterical attack in which she lay for a time in a trance-like state, unable to speak but conscious of surroundings. Since then, after each menstrual period, she has had severe headaches, vomiting, hysterical spells in which she lies as if in a faint, but conscious of everything. She complains of being easily tired, of inability to concentrate her thoughts, and of numbness and tingling in her fingers. She is habitually constipated and passes but little urine. She complains bitterly of headache and dizziness, and fears she will become insane. Her emotions are easily aroused and she cries at the least provocation.

Patient is 25 pounds over weight. Her heart is inclined to tachycardia. Breathing is sighing at times. Abdomen is pendulous, flabby, and fat. Otherwise her condition is normal except certain stigmata of hysteria, such as hypesthesia of half her body, irregularity of color fields, diplopia at times.

Treatment was directed against her constipation and likewise against the hysterical element that was so prominent in her case; she promptly recovered from the latter. In time the proper hygiene of her intestinal tract and kidney elimination bore fruit, and she become free from the neurasthenic element as well.

CASE VIII.—Mrs. W., aged thirty-six years. Both parents were rheumatic. Her father had heart and kidney disease.

She has been married seventeen years. Seven years ago she was operated upon for the removal of one ovary. The same year she developed asthma which has been present at times ever since. She has always been of a nervous, worrying disposition.

Complains of asthma, mental depression, and fits of crying. She is easily tired. She has worried a great deal about her husband's health. Her bowels are constipated and she is passing only thirty-two ounces of urine in twenty-four hours.

Her symptoms are not attended with much distress, but are gradually becoming more severe. Heart and lungs are normal. No abnormalities of position of viscera noted. Abdomen is pendulous. There is some asymmetry noted. There are no gross stigmata of hysteria.

Examination of urine at the Philadelphia Clinical Laboratory shows indican very high; otherwise normal.

After treatment that was principally dietetic and hydrotherapeutic together with massage and regulated exercises, she became much better. Nine months later examination of her urine showed a normal output of indican.

In the above cases certain points stand out prominently. The first is hereditary taint and a neuropathic susceptibility. The second is the symptom of fear in its various phases—so common in intestinal diseases. The third and most important as far as treatment is concerned, is the constant condition of intestinal putrefaction that has had to be covercome before any relief was achieved; this was in many of the cases associated with insufficient elimination of urine. In cases of this kind there is often a hysterical taint engrafted on the original trouble that must be considered in treatment.

THE INDICAN REACTION AS EVIDENCE OF ENTEROGENIC INTOXICATION.

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IT is conceded that there is great danger in removing any symptom from its clinical surroundings for the purpose of weighing its intrinsic worth; but if there is sufficient evidence to formulate working rules, that danger should not prevent us from seeking to study limitations. The danger is probably no greater with any other clinical test than it is with the Jaffé or Obermeyer reaction in the urine. Indican is present in excess so frequently, not only as

evidence of a primary pathological process, but of a complicating condition, that its very frequency leads to misinterpretation.

Further reason for this inquiry is found in the fact that the indican reaction is really the only test available to the general practitioner as an index of enterogenic intoxication.[1] Partition estimations of the sulphuric-ether excretion in twenty-four hours may be exceedingly valuable, but such work is ordinarily out of reach. The same may be said of bacteriological or chemical examination of the feces. Estimations of sulphur intake with the food and its ratio with the sulphur of the urine are usually limited to institutions for medical research. Furthermore, a few observations have been published which make one feel that the simple indican test is a better clinical indication of intoxication arising from putrefactive processes in the intestine than other more refined methods; or at least is a better indicator of the capacity of the organism to assimilate nitrogenous food.

The following evidence is presented in the hope that it will stimulate interest and confidence in the Jaffé and Obermeyer tests. It is drawn largely from laboratory observations, because of the general pessimism which exists among many such workers as to the value of the indican reaction. Precision tests should be devised as soon as possible, but in the meantime walking with a crooked stick is better than stumbling with no stick at all. No attempt will be made to discuss the interpretation of excessive urinary indican. That is a matter of clinical experience. But if some knowledge can be gained of the origin and the difficulties which befall the precursors of indican in the organism, the interpretation will naturally tend toward truth.

The work of Ellinger[2] has removed any doubt which may have existed in the past as to the source of urinary indican. In those starving animals which could be prevented from eating their own feces, the indican reaction in the urine subsided and disappeared as death approached. It could therefore not be formed during the course of proteid metabolism. Gentzen[3] has proved that the injection of skatol-amino-acetic acid (the mother substance of indol) into the cecum was followed by an abundant indican excretion. If ingested with the food, or injected into the rectum, it was metabolized. Increased excretion of indican secondary to putrid empyema

[1] The terminology is unfortunately ambiguous. Intoxications arising from saccharobutyric fermentations in the intestines are not considered, but only those arising from proteid putrefaction.

[2] Zeit. f. physiol. Chemie, 1903, xxxix, 44. Many animal experiments have been undertaken for the purpose of working out problems connected with the etiology and effects of gastrointestinal intoxications. It seems as if too much importance as applied to man has been attached to some of them, particularly to those which involve conditions in the intestine. The habits of different animals have marked effects on intestinal putrefactive processes. Dogs are very apt to excrete large quantities of indican. This fact is probably traceable to eating filthy food and their fondness for meats. Allowance should be made for these differences in habit and for the lack of exercise which most experimental animals experience.

[3] Ellinger and Gentzen, Hofmeister's Beiträge, 1903, iv, 171.

or putrefying fœtus *in utero* is of rare occurrence. It is not in this class of cases that the reaction is of assistance in diagnosis, while by no known means can the intestine as a source be excluded. Abscess does not cause it (Beckmann).[4]

The formation of indol is, therefore, limited to the intestine, and it appears during the course of proteid putrefaction and as the result of microbic activity. The conditions which promote its formation are pertinent points for inquiry in considering the excretion of indican in the urine.

CHEMICAL CONSIDERATIONS. On the rough average, the aromatic nucleus of the proteid molecule—of which preformed indol is a part—amounts to about 5 per cent. In some proteids it is absent (gelatin), while in others it may exceed that amount. If it could be assumed that disintegration of the proteid molecule under bacterial influences always took place along fixed lines, and therefore always led to the same end-products, the problem would be much simplified. It would be possible to work out with mathematical accuracy the amount of indol which could be formed from any given proteid.[5] This assumption is not possible. Under some circumstances no indol may be formed, while under different conditions indol may be formed to some extent at the expense of other end-products.

For reasons which will appear later, there are objections in applying these laboratory results too literally to putrefaction in the intestine. In case the end-products of bacterial digestion are not removed, as would obtain in the intestine (even if mixed fecal bacteria are employed and other conditions are imitated), there is a tendency for the activity of the bacteria to be limited as the end-products increase in concentration. Bacterial autolysis is undoubtedly favored; or further activity is expended in dissociation of substances already formed into the simplest end-products.

Indol formation, except in grossly pathological states, is confined to the cecum and the ascending and transverse colons. Not more than 14 per cent. of ingested nitrogen passes the ileocecal valve in approximately healthy conditions (Mayfadyen, Nencki, and Sieber[6]). This includes nitrogen in the form of true excrementitious matter derived from the mucosa above and the secretion of glandular organs contributory thereto. From 6 to 10 per cent. passes the rectum in the feces. It[7] is assumable that nitorgen in almost any form

[4] St. Petersb. med. Woch., 1894, Nr. 28.

[5] Vide article by Fr. Müller in v. Leyden's Handbuch der Ernähungstherap., 1903, i, 162.

[6] Journ. Anat. and Physiol., 1890–91, xxv, 421. These figures must be taken with a great deal of latitude, as the conditions are probably varying. There is no more certain way of causing increase in indican excretion than by excessive ingestion of proteid food. On the other hand, this does not cause a *proportionate* increase in fecal nitrogen. The difference probably falls prey to bacterial activity as soon as the assimilation limit is reached.

[7] With regard to fecal nitrogen, it may be said that if a complete partition could be made in health and disease, it would give us much information as to the intensity of bacterial processes above. This would *not* be a measure of the intoxication. Only that part which is absorbed is concerned in the latter process. For this reason intestinal putrefaction in the maintenance of nitrogen balance is ignored, as the factors tend to balance each other to a small fraction.

may be utilized by microörganisms, but only certain forms of proteid will yield indol under any circumstances. Many forms of nitrogenous matter reach the ileocecal valve, but only a few contain preformed indol, or are capable of yielding it. These factors would certainly affect the indican output, to what extent it is most difficult to estimate, except that the evidence seems to favor the view that as an end-product there would be a minimum production of indol.

Bacterial Considerations. According to the view originally advanced by Bienstock[8] and now generally accepted, Bacillus coli and other facultative aërobes which are "normal" inhabitants of the bowel, are regarded as defensive organisms as opposed to certain obligatory anaërobes (Bacillus aërogenes capsulatus, Bacillus bifidus, etc.), which are regarded as putrefactive organisms and capable of originating pathological putrefactive changes in the intestinal contents. Bacillus coli and its associates exist in a vegetative state, limited in their activities by a peculiar combination of circumstances and by active defences of the host. They expend most of their energy normally in propagation. They cannot attack native proteid, but do form indol. On the other hand, the obligatory anaërobes do disintegrate native proteid at the expense of some time and effort, but they are unable ordinarily to liberate indol (Rettger[9]). Putrefaction of proteid without anaërobes is impossible. Therefore indol formation results from the intervention of Bacillus coli and related forms, acting either on the products of anaërobic activity or on the products of normal proteolytic digestion which have passed along unabsorbed.[10] A good deal of stress is placed on this last point for this reason: ordinary attacks of biliousness in young people, accompanied by large indican excretion and resulting from overeating, presupposes, unless the above alternative be accepted, a complete and sudden change in the bacterial content of the bowel. This seems inconceivable, particularly as the condition yields to proper treatment in a few hours, which in turn presupposes quite as sudden a reversal in bacterial relations to the normal.[11] It is also possible on this hypothesis to account for other discrepancies in the theory of enterogenic intoxication, of which space forbids further consideration.[12]

[8] Arch. f. Hyg., 1901, xxxix, 390.

[9] Jour. Biol. Chem., 1906, ii, 71.

[10] There is some reason for believing that the production of indol as an end-product is dependent on the integrity of the pancreatic secretion, or at least on the ability of the gastrointestinal secretions, to hydrolyze to the peptone stage.

[11] Vide Herter, Jour. Biol. Chem., 1906, ii, 65, §12.

[12] The products of proteolytic digestion—the carbon nuclei and particularly peptones—are themselves capable of causing intense inflammatory disturbances in the enteron, particularly in concentrated form. This is so well known among laboratory workers that certain types of experimental work cannot be carried on without strictest precautions. There is also a very sharp limit to the capacity of the small intestine to assimilate and rehabilitate carbon nuclei into body proteid. This is especially true if the mucosa is in a state of catarrhal inflammation.

VARIATIONS IN THE ABSORPTIVE POWER OF THE INTESTINE FOR INDOL. It is by no means certain that under strictly normal conditions the mucosa of the large intestine absorbs any considerable quantity of indol, even if elaborated. That it does so in a vast majority of adults is no argument that it is normal in the strictest sense of the term.[13] Some light is thrown on this question by estimating the indol which appears in the feces and comparing it with the amount of indican excreted by the kidneys. Baumstark[14] has made such determinations and found that, with one exception, fecal indol was much greater than urinary indican. This occurred in presumably healthy persons and in those suffering from disease.[15] It would seem as if this variability in absorption is the first defence of the organism against the products of intestinal putrefaction. For this reason, if for no other, any method of diagnosticating the presence of enterogenic intoxication by estimating proteid purefactive products in the feces is fallacious.

What has been said of the absorption of indol applies more particularly to that part of the intestinal tract which lies below the ileocecal valve—to that part in which "normal" putrefaction takes place. In admittedly pathological conditions (such as ileus) putrefaction and the formation of indol is very rapid, and the appearance of indican in the urine is prompt and excessive (Ellinger and Prutz[16]). One can hardly refrain from saying that these very differences in absorption contribute in a striking manner to the value of the indican reaction as indicative of intestinal putrefaction, for in that part of the intestine in which indol is most readily absorbed its presence is certainly pathological.

The detoxication of indol—by union with sulphuric acid and potassium subsequent to oxidation—occurs mainly in the liver. Indoxylpotassium sulphate is, of course, indican. Any combination which the liver may make with glycuronic acid instead of sulphuric acid represents a distinct loss to the urinary reaction for indican. This union is not excessive, and probably happens only when the production of sulphuric acid esters is in great excess and the supply of sulphuric acid ions is limited (Schmiedeberg[17]).

Between the intestinal tract and the urine there is a great loss of

[13] One would hardly argue that because 95 per cent. of those people who come to autopsy after the fiftieth year, show evidence of nephritis, that this is a normal condition.

[14] Münch. med. Woch., 1903, 722. Observations on the relation of fecal indol to urinary indican in healthy children would probably solve this problem.

[15] Baumstark's exception was a girl, aged eleven years, suffering from septic peritonitis. It is of interest to note with this that Herter (Jour. Biol. Chem., 1906, ii, 6) in cases of pernicious anemia, made similar comparisons. He found—judging from the table given—that there was no parallel between the two. In both these conditions the intestinal tract is in an abnormal condition. The inference is plain. Furthermore, indol elaborated in the small intestine would be much less liable to reach the feces owing to the fact that the absorptive power of the small intestine for indol is very great even in health.

[16] Zeit. f. Physiol. Chem., 1903, xxxviii, 399.

[17] Arch. f. exp. Path. u. Pharm., 1881, xiv, 288.

indol, totaling to from 40 per cent. to 75 per cent. (Jaffé, Wang, Ellinger[18]). What is the fate of this lost indol? Herter and Wakeman[19] found that the "living cells of the body and especially the hepatic and renal cells and the epitheliated cells of the intestinal tract have the power of absorbing considerable quantities of indol, as well as phenol, and tying them loosely in such a way that these bodies cannot be recovered by distillation." The proper interpretation of this phenomenon may lie in two directions. There is a possibility that a certain portion of indol in an organism with vigorous oxidative capacity is disintegrated into primary carbon and nitrogen groupings, to be recombined in other ways or eliminated as simple end products and thus lost.[20] On the other hand, have we here another manifestation of defensive action on the part of the organism in that the cells at large are able to store indol or its derivatives—withdrawing it from circulation—until such time as circumstances and capacity permit of release in detoxicized form? Perhaps both elements play a part. The point of interest is the loss of indol in transit. It seems quite certain that the organism may change this factor within limits at different times.[21]

That normal bile contains both ethereal sulphates and glycuronates in traces has been noted by Hammarsten. Whether in pathological states this amount can be materially increased, thus delaying or preventing excretion by the kidney, is not known.

We are uninformed as to the capacity of the kidney for the elimination of indican presented to it by the blood stream. In general, renal epithelium takes kindly to sulphur in any form, for even in renal disease the elimination of sulphur parallels that of nitrogen. Sulphates are excreted in the same ratio as urea, until the rate of elimination reaches a certain excessive point when the kidney refuses to maintain the ratio (Cushney).[22] However, this may not apply to ethereal sulphates. If one may reason from analogy, there may be occasions in the course of a nephritis when, by reason of great excess, portions of indican or its precursors are retained in the blood or there may be variations in the rate of excretion. Possibly the regulating mechanism lies further back and the excess is retained or fixed in the body cells until the serum concentration has sufficiently

[18] Jaffe, Virchow's Archiv, 1877, lxx, 78. Wang, Ziet. f. physiol. Chemie, 1899, 27, 557. Ellinger and Gentzer, Hofmeister's Beiträge, 1904, iv, 171.

[19] Jour. Exp. Med., 1899, iv, 307.

[20] The benzene ring is exceedingly refractory to disintegration, and it is broken up by the organism only at the expense of considerable energy. In considering the toxicity of indol or any of its derivitives, this point must be kept in mind, that excessive quantities of indol thrown into the circulation for a long period of time may impair oxidative capacity. It could hardly be claimed, for instance, that one three-finger glass of whiskey is "toxic." However, a man who made a practice of taking three such drinks a day for ten years will present plenty of evidence of it even if considerable carbohydrate has been spared in the process.

[21] Part of this loss may undoubtedly be accounted for by technical difficulties in the recovery of all the indican excreted.

[22] Jour. of Physiol., 1901–02, xxvii, 429.

diminished to permit its release. Brain tissue has this power (Herter).

Jaffé's original estimate[23] of the "normal" excretion of indican for twenty-four hours was based on observations made on eight presumably healthy people. One excreted no indican at all, and one excreted over 19 mg. The average for the eight was 6.6 mg. It was therefore concluded that the normal output was from 5 to 20 mg. per diem. But the difficulty has been to determine just what the word "normal" should signify. If by it is meant "ideally normal," Jaffé is unquestionably wrong—there should be no indican excretion. I am satisfied of the truth of this for two reasons. The urines of heathy children rarely contain appreciable quantities of indican. The same can usually be said of those adults of middle life whose organisms are in good condition and particularly those whose habits as to the use of meat are moderate. Chittenden[24] found during the course of his dietary experiments that the urinary ethereal sulphates gradually disappeared. Fermentative and putrefactive properties of the feces diminished, as "Fletcherizing" subjects naturally ingested less meat, according to observations made by Fisher.[25]

It seems quite possible that the organism might excrete 20 mg. indican daily and not suffer symptoms thereby, provided the oxidative capacity is high and the process does not extend over too long a time. Either of these provisions violated, and the subject will straightway seek a doctor. On the other hand, less than 20 mg. may suggest the source of subjective symptoms if the oxidative capacity is low. In this class of cases the interpretation of the reaction really resolves itself into an inquiry concerning the ability of the patient to detoxicize products of intestinal putrefaction which may be arising at the time.

But if we assume with Jaffé that 20 mg. is about the limit of normal excretion, there is in this a basis for calculation with Obermeyer's reaction,[26] which may be depended upon for clinical purposes;

[23] Pflüger's Archiv, 1870, iii, 448.

[24] "Physiological Economy in Nutrition."

[25] Trans. of the Conn. Acad. of Arts and Sci., 1907, xiii, 13.

[26] Until very recently Obermeyer's reagent was used for determining quantitative estimations of indican, but it does not yield constant results. There may be as much as 20 per cent. loss (Ellinger, Zeit. f. physiol. Chemie., 1903, xxxviii, 178). This loss is *liable* to be greater with the Jaffé reaction as applied to qualitative work. There is a great temptation to use too much of the oxidizing agent (potassium permanganate, etc.), which further oxidizes and decolorizes the indigo-blue. The agents should be fresh. When the urine, acid, and oxidizing agent are mixed, they should commingle at the same time, after the manner suggested by Emerson, and not as usually done by mixing two and adding a third. After the mixture is made with either reaction, it should be turned against the finger once and allowed to stand for five minutes. Heating to blood heat before adding the chloroform will also assist. According to Ellinger, there is a loss of 10 per cent. even under the most favorable circumstances. If quantitative estimations are desired, it is well to remember that in urines with high indican content, variations in the quantity of oxidizing reagent and chloroform are necessary to insure maximum color. The better method is to dilute the urine with known quantities of water in the presence of an excess.

20 mg. of indican in 1000 c.c. of urine will yield with Obermeyer's reagent, as ordinarily practised in the laboratory, an index of somewhere between 50 and 75 on Folin's scale.[27] An index of 100 or several times that amount is not uncommon. Heeding all factors given above as necessary for the production of such indices, it must be evident that such a symptom and the intoxication it represents is worthy of very serious consideration, particularly if there is any tendency for it to continue, or if it is accompanied by evidences of digestive, hepatic, or renal disease. Perhaps the following will present some working rules until something better is devised:

1. Urinary indican is a product of intestinal putrefaction. There may be putrefaction without the production of indol, but there cannot be indicanuria without putrefaction.

2. A maximum excretion of indican, that is, an amount which on Folin's scale gives an index of 100 or over, may be safely relied upon to indicate excessive intestinal putrefaction, and especially the intoxication arising therefrom.

3. A maximum reaction which gives an index under 100 may be significant, but its interpretation should be strictly guarded by the general condition of the patient, that is, by the oxidizing and excretory capacity.

4. A heavy indican reaction which markedly subsides under treatment undoubtedly indicates a lessening intoxication, but minor variations in the color index have no significance at present.

5. No interpretation can be placed on a negative reaction, too many unsolved factors entering into the problem.

These are the rules which have been followed in my practice for some time, and which I have found very satisfactory. It is conceded that they are not perfect, but they furnish a practical working hypothesis. It must also be stated that in singling out one of many putrefactive products it has been impossible to do other than take a very narrow view of the subject as a whole. There is much more evidence on the condition of which indicanuria is an indication, but it cannot be presented here. Due allowance must be made for these considerations if the principles herein are to be applied to concrete cases in which intoxication arising from intestinal putrefaction is suspected.

[27] Otto Folin (Amer. Jour. of Physiol., March, 1905), as a matter of convenience, in reporting some urinary examinations, used a scale based on the color of Fehling's solution. A urine which precipitated a color with Obermeyer's reagent equal to Fehling's solution was said to have an indican index of 100. Exact determinations were obtained by diluting either the urine or the Fehling's solution according to the index, whether above or below 100.

THE TREATMENT OF CHRONIC STENOSIS OF THE LARYNX AND TRACHEA.

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In a previous article upon this subject[1] a plan of treatment which seemed capable of success was given in detail, and 23 illustrative cases were described. As enough time had not then elapsed to make the results conclusive, and as now all the patients who were then living have recovered, it seems proper to review the procedures by which success was obtained. In those 23 cases at the time of the last report, there had been 3 deaths and 2 failures to cure; the misfortunes I think were largely dependent upon my inexperience. All of the remaining 18 patients are practically well, and all but one have good or perfect voices. Two, however, of these 18 are still wearing intubation tubes. One of them is a practising physician, who has acquired the somewhat remarkable accomplishment of intubating and extubating himself, and cannot be induced to wear the tube continuously and long enough permanently to cure his cicatrix, which at constantly longer intervals shows signs of recontraction, and from time to time requires some dilatation. The second patient has a cicatrix just below the vocal cords, and though a trial has shown that it will not now shrink enough appreciably to obstruct respiration, it interferes so much with the cords as to permit only whispering speech, to overcome which disability its complete obliteration by pressure is being attempted. With these two exceptions a practically perfect result can be announced for 18 out of the total of 23 patients.

The causes of obstruction in the larynx or trachea may be briefly recapitulated as follows: (1) Obstruction or compression of the respiratory tract by neoplasms, of which the papillomas are the most common, and exuberant granulations rare. (2) Cicatrices of traumatic, operative, or inflammatory origin. (3) Hypertrophic subglottic laryngitis. (4) Atrophy (of disuse) of the laryngeal abductors in "retained tubes" and tracheal cannula cases. (5) Other less common causes of obstruction might be cited, but there is no necessity of multiplying the classification, as the cases must evidently be either entirely irremediable or else possible of treatment by operation or a combination of the operation and subsequent dilatation methods here advocated.

This work was begun with the object of relieving some apparently hopeless cases of retained intubation tubes and tracheal cannulæ which were sequels of diphtheria, and with the experience thus gained

[1] Amer. Jour. Med. Sci., November, 1905.

was extended to the treatment of the more strictly surgical conditions of fibrous strictures and of neoplasms. Hence this order will be more or less followed below, as it showed the way to overcome many obscure difficulties and dangers.

Statistics have demonstrated that about 1 per cent. of the patients intubated for laryngeal diphtheria will subsequently be unable for an indefinite period to breathe without the assistance of the tube or a tracheotomy. As such cases are at the best (or worst) not numerous, and as autopsies are often not obtained, the true pathology of the condition is not generally recognized or is disputed.

In my previous article there were cited in detail 15 cases of "retained tube" following laryngeal diphtheria. In this group there were 3 autopsies, and in addition the larynx and trachea of 5 cases were carefully inspected by thyrotomies and tracheotomies. In these 8 patients the pathological condition was thus verified by sight and touch, and in 3 of them was studied under the microscope and found to be a chronic hypertrophic laryngitis, a combination of an exudative and productive inflammation causing a vascular, œdematous thickening of all the intralaryngeal soft parts sufficient to occlude completely the respiratory tract from the false cords to the lower border of the cricoid cartilage or the first or second ring of the trachea. In the other 7 cases the clinical picture was so similar, as was also the sensation communicated to the operator's hand during intubation, that there can be no reasonable doubt of the chief primary cause of obstruction in these patients, all of whom had to be intubated for laryngeal diphtheria, and thereafter could not breath without the instrument. Ulceration and cicatrices may develop at the same time or later (generally as the result of tracheotomy or other operation), but a cicatrix as the original source of obstruction after diphtheria only occurred twice in my cases, and then the diagnosis was doubtful. Exuberant granulations as a cause of obstruction occurred but once in the postdiphtheritic cases, and then were secondary to an intubation for hypertrophic laryngitis. Spasm or cramp of the adductors simulating an hypertrophic laryngitis occurred three times.

In considering the reasons for the stenosis in these retained tube cases following diphtheria the above experience may be considered a fair example of the average: hypertrophic laryngitis, spasm of the adductors, cicatrices, and exuberant granulations occur in about this order of frequency; the difficulty in the great majority of cases is, however, a chronic exudative and productive inflammation of the intralaryngeal soft parts; in other words, a chronic hypertrophic subglottic laryngitis. The condition was recognized long before the invention of intubation, when tracheotomy was the only remedy possible for severe croup, and hence it is not fair to ascribe it to faulty technique in the "tubage." The chronic thickening may have something to do with traumatism inherent in wearing a tube,

but the individual operator should not be held responsible for what is properly a danger inseparable from a life-saving procedure; and this danger is comparatively slight when one considers that it only occurs in about 1 per cent of the cases.

What I have called "adductor spasm" as a cause of stenosis is not generally recognized, but is a condition of serious moment in patients who have worn a tube for a long period of time. This instrument, particularly the "special" tube recommended below, places the abductors or openers of the larynx entirely at rest for weeks or months, while with every effort in coughing or clearing the throat the adductors are brought into use; thus one set of muscles, the closeers of the larynx, should, theroetically at least, hypertrophy, and the other set, or openers, atrophy. I have never been able to verify the condition by autopsy, but the treatment necessary to overcome it corresponds so exactly with the hypothesis that I have no doubt of the accuracy of the description, and unless it is borne in mind some of these cases will be given up as hopeless instances of intralaryngeal hypertrophy or abductor paralysis, or what not, and condemned to a permanent tracheotomy. Localized obstructing paralysis may be possible, but from a study of the literature and from observation in a considerable experience I believe it must be extremely rare.

The differential diagnosis of the causes which may necessitate the retention of an intubation tube (or a tracheal cannula) after laryngeal diphtheria for weeks or months is not difficult. The common hypertrophic laryngitis usually produces complete obstruction, which is manifest immediately or very rapidly after the removal of the artificial aid to respiration. A cicatrix, on the other hand, does not immediately occlude the lumen of the passage when the dilating instrument is removed, but it contracts gradually in the course of days; and more distinctive still is its resistance to instrumentation. The soft œdematous tissues of an hypertrophic laryngitis require no force in the passage of the proper sized tube or urethral sound, while for the introduction of an instrument through a fibrous structure some pressure has to be exerted. Furthermore, the frequently dangerous accident of auto-extubation, especially when it occurs repeatedly and without apparent provocation, generally accompanies hypertrophic laryngitis, and seems to be largely caused by it. It often occurs in the presence of both hypertrophic and cicatricial stenosis, but very rarely when the condition is purely cicatricial.

During the manipulations exuberant granulations, which are very seldom the true cause of the obstruction, should be seen or felt; they can be generally found around a tracheal fistula or at the upper or lower end of a tube or cannula, and should reveal their presence by difficulty in inspiration rather than expiration or in the reverse state of affairs. They are friable and bleed easily, and if ordinary care is employed cannot readily be overlooked.

The condition of adductor spasm occurs only in those patients who have worn a tube, or much less commonly a cannula, for weeks or months, and so closely simulates hypertrophic laryngitis in the immediate and complete obstruction as to be indistinguishable from it without the aid of general anesthesia. Then, when consciousness and the pharyngeal reflexes are abolished, the stenosis, which previously had been complete, will be found to have disappeared and natural respiration will be evident. Occasionally the spasm will not appear for several hours or days after extubation or removal of the cannula, and then will occur suddenly with a stridulous inspiration following a cough or some excitement or slight exertion. There is apparently a lack of coördinate control as well as an overabundance of strength in one group of muscles and a diminished strength in the abductors. The only way of rectifying the possibly fatal discrepancy is to build up the strength of the openers of the larynx and to allow the patient time and opportunity to regain control of them and become conscious of that power.

The principle which I have found successful in the treatment of hypertrophic laryngitis and in cicatrices of the upper part of the respiratory tract involves only long continued and constant dilatation, which stretches the contracted tissues to the utmost limit they will bear without sloughing and up to the largest normal caliber of the passage in question, and so overcomes the obstruction by producing a permanent pressure atrophy in the affected parts. A few of the less severe cases of retained tube due to hypertrophic laryngitis after diphtheria will recover under the application of astringents or the use of a tube as small as can be safely worn, with the object of minimizing traumatism; occasionally tracheotomy has been reported as intentionally or accidentally succeeding in the same way. But unless there is some distinct indication for the operation, such as dangerous auto-extubation, it seems hardly justifiable to add to the already bad enough condition the probabilities of a cicatrix or tracheal deformity which so often follows the long continued use of the curved cannula. And when it is remembered that hypertrophic laryngitis was a not uncommon cause of obstruction after long retention of the cannula in the pre-intubation days, one should hesitate still more at the infliction of such additional risks.

To accomplish the continued dilatation and consequent pressure atrophy necessary to overcome the obstruction of either a hypertrophic laryngitis or a cicatrix a properly made O'Dwyer intubation tube is almost a perfect instrument. It can be worn without harm to the patient for months or years, and I have observed such patients with the tubes in place to pass successfully through measles, pneumonia, typhoid fever, and even whooping-cough; indeed it does not seem appreciably to increase the gravity of the prognosis of these affections. The tube, as is well known, possesses a head which rests on and in the aryteno-epiglotic folds, a neck constricted from side to

side to fit between the vocal cords within the glottis, and below a retaining swell which expands to occupy the space at and below the cricoid cartilage. The lower end in the children's sizes is made longer than in those used for adults, as it was designed to extend well beyond the false membrane of a laryngeal diphtheria. The tip is again constricted from side to side to pass easily through the glottis. The "normal" tubes were designed by Dr. O'Dwyer from casts and measurements to fit somewhat loosely the average larynx and exert the least possible pressure upon acutely inflamed tissues compatible with retention of the instrument. When this stage of the disease has passed and been succeeded by a chronic thickening or a cicatrix, the tube, to obtain the pressure necessary to produce an atrophy of the abnormal thickening, must be modified into a "special" instrument for each case. That is, with the patient under deep general anesthesia, a number of normal tubes are passed through the larynx until one is found in which the retaining swell distends the constriction to the limit the operator believes it will bear without sloughing. If the difficulty is dependent solely upon a soft hypertorphy, the diameter of the retaining swell will be regulated largely by the diameter of the passage within the cricoid cartilage. Inasmuch, however, as the retaining swell lies, when the tube is in place, just below this point, where the trachea is capable of considerable distention without damage, quite a little force in the test-tubage is permissible. Having thus ascertained the largest possible diameter of the larnyx a "special" tube is made of the length of the normal tube suitable for the age and development of the patient, but with a retaining swell having the same diameter as that of the trial tube. The neck of this "special" tube, however, to exert pressure upon the hypertrophied intralaryngeal tissues, should be as large as possible, but its transverse diameter must be $\frac{3}{32}$ of an inch smaller than the retaining swell, or it will certainly be coughed out. Also the head must have a diameter $\frac{6}{32}$ of an inch greater than the retaining swell to prevent the instrument from slipping within the larynx. These measurements apply to the ordinary hard rubber tube, but when the heavier metal tube is employed the diameter of the head should exceed that of the retaining swell by $\frac{7}{32}$ or even $\frac{8}{32}$ of an inch, especially for adults. For very small children these measurements can be changed from thirty-seconds to sixty-fourths of an inch. It must be borne in mind that a difference of $\frac{3}{32}$ of an inch in the transverse diameters of the neck and retaining swell is the very least which experience has demonstrated is sufficient to keep the tube in place, and many patients will require a tube with the neck $\frac{4}{32}$ or even $\frac{5}{32}$ of an inch less in diameter than the retaining swell. Only a trial will show what is necessary. But the neck of the special tube must be considerably larger than that of the normal, or a failure to cure will probably result.

When auto-extubation complicates the difficulties, and it may be a very dangerous complication if assistance is not constantly and

immediately available, tracheotomy is almost always necessary. Some patients with hypertrophic laryngitis will cough up any tube which can be inserted, and, unless it is quickly replaced, will strangle. Under these circumstances it is generally essential to provide an opening in the trachea, though there can be no expectation of thus accomplishing a cure of the stenosis. Indeed, it will probably only add a cicatrix or a deformity in the shape of an obstructing "spur" in the posterior wall of the trachea if the cannula is worn long enough. The operation should be performed carefully in the median line well below the cricoid cartilage, under full anesthesia, with the tube in place. At the same time opportunity is thus given to obtain the necessary measurements for making what I have described as a "plugged" or "clamped tube." This is a "special" intubation tube with the neck (or lower end if there is a cicatrix low down) enlarged to exert pressure upon the stenosing area. The "clamp" or "plug" portion of the tube is a removable attachment designed to fasten to the tube at right angles through the tracheal fistula after the tube is inserted, and thus prevent its displacement. The larger tubes will support a "plug" or a cylindrical piece of hard rubber, which is long enough so that when in place its outer end extends through the fistula about an inch beyond the surface of the neck, to allow easy manipulation, and its inner end, perforated to correspond to the lumen of the intubation tube, screws into a hole in the anterior wall of the intubation tube. The "clamp" is employed for smaller instruments, and is made, like a pair of obstetrical forceps, of two little silver blades, which are passed through the fistula into grooves on the sides of the tube and held there by a "collar" screwed down around the shanks.

Of these two attachments, the plug is probably better for large or for metal tubes, and the clamp for smaller instruments and those made of rubber. The clamp is also better in that it can be made small enough not to press apart appreciably the severed anterior segments of the trachea, and thus, by preventing contraction in the soft posterior wall, there will not be so much danger of narrowing the caliber of the passage when the instrument is removed. A tube with either of these attachments cannot be coughed out, and can be worn for an indefinite period without harm, and in all respects is infinitely better than the ordinary cannula. If, therefore, a patient with a hypertrophic or cicatricial stenosis of the upper air passage has to be tracheotomized, one of these instruments should be inserted as soon afterward as it can be obtained, and continued in place as advised for the usual special tube. Neither a plugged nor a clamped tube should, however, be employed unless there is some good reason for it, as the tracheal fistula gradually, of course, becomes permanent and presents its own difficulties, although these may be offset by the ease with which after removal of the tube any adductor spasm can be met.

The usual intubation tube is made of vulcanized rubber, which is an ideal material, as I have never known it to clog and it is practically indestructible. It does, however, have to be removed and cleaned about once in a month or six weeks, as the head generally becomes foul, and this decomposing matter might cause sepsis or possibly obstruct the lumen. But occasionally in cases of hypertrophic laryngitis the special rubber instrument, even after months of continuous use, will seem to produce no improvement. Then by changing to a similarly made metal tube the desired result will generally be obtained. I am unable to explain this except that the metal tube is heavier and so may exert more pressure; or there may be some electrolytic effect, as it is constructed of bronze or brass heavily plated with gold, and so consists of at least two metals which are in contact with moisture. Whatever reason may be given, the metal tube will often succeed when the rubber has seemed to fail, but the latter should always be tried first, as in the average case it is sufficient and safer. The metal tube soon becomes rough with a presumably calcareous deposit, which catches the secretions, and in at least one of my cases thus produced asphyxia. After wearing such an instrument, preferably of hard rubber, for months, it is very necessary to consider the adductor spasm both for diagnosis and treatment. When a cure ought to have been obtained the conditions may seem entirely unchanged, and the true state of affairs can only be ascertained by general anesthesia. Under ether, then, about once in every three or four months the tube should be carefully removed and the respiration observed. If it is still completely or partially obstructed, the dilatation must be resumed. But if the breathing is natural and easy, then spasm may be confidently diagnosticated, especially if in the first effort at coughing, as consciousness comes back, the dyspnœa recurs. To overcome this there must be inserted for several days or weeks a tube with a head and retaining swell and length like the previously worn dilating instrument, but with a neck as small as possible, in order that motion to some extent can occur in the openers of the larynx. If the dyspnœa makes its appearance slowly during the course of hours or days, it is not due to adductor spasm, and there is no escape from the previous treatment. When enough time, or some one or two months have passed with the very small necked tube in the larynx, and the faulty muscles may be considered to have regained their strength and the patient his power to innervate them, the small tube is removed under ether and a little morphine is administered to allay spasm and nervousness. For the first few hours, however, the attendant and the patient will suffer much anxiety, as frequent attacks of stridulous inspiration and more or less dyspnœa will occur during the readjustment to new conditions. But with patience and the assistance of antispasmodics a day or two will suffice to overcome all such difficulties.

A fibrous stricture is managed in exactly the same way, with the

exception of the primary dilatation in case the stenosis is too tight to admit a tube. Under these conditions there is already a tracheal fistula, or one is urgently needed, and through this under general anesthesia urethral sounds are passed up and down until considerable resistance is encountered. Then measurements are taken upon an ordinary tube and a "special" one is constructed having its specially enlarged part placed to correspond with the location of the stricture and with regard to the normal diameter of the air passage, which is capable of much stretching without damage except within the cricoid ring. When the latter has been cut in a hasty previous operation there seems to be little danger of sloughing under almost any reasonable pressure, but if it is intact, such an accident is conceivable, and actually occurred in a case cited by Dr. O'Dwyer. Sometimes in very dense strictures a small tube can be inserted for a week or two, and afterward a larger size. But no stricture should be considered hopeless provided any trace of mucous membrane remains, as I have had experience in at least one such case in which the passage through the larynx had to be opened by a careful dissection very much like an impermeable stricture of the urethra. A small tube was then inserted and the wound partially closed, and later larger instruments were employed, and eventually an almost perfect result was obtained.

As regards the prognosis and duration of treatment in patients with hypertrophic laryngitis or cicatricial stenosis, the statistics of the 23 cases previously reported furnish much information. One case of hypertrophic laryngitis (Case I), the only one in the series which seemed to have occurred spontaneously and without a previous diphtheria, recovered permanently at the end of about four weeks of dilating tubage. Another of cicatricial stenosis (Case XXIII of Dr. Simpson's, previously reported as a failure) required forty-nine months, or a little over four years, of continuous intubation, and during the latter two of these four years the tube was not once removed from the larynx. It has now been out for over six months, and there is no sign of recontraction and the voice is returning. Cases X, XII, and XIII, the most discouraging of the series of instances of hypertrophic laryngitis complicated by cicatrices due to tracheotomies and explorations through the thyroid cartilage, required respectively about six, three, and five years of dilating tubage to effect a cure, which now may be considered permanent, as they have remained for nearly two years without any sign of difficulty and all have good voices. Cases X and XIII would have recovered sooner if I had had less impatience and more experience, for as soon as they seemed well their tubes were removed, with the result that the cicatrices recontracted. A fibrous stricture apparently requires at least two years of continuous dilatation to the utmost limit of the normal lumen of the respiratory tract before it can be considered as permanently overcome. Even then it may gradually

reappear, and if it does another six months or a year of continuous tubage must be endured, but there is a reasonable certainty, as proved by experience, of thus gaining an ultimate cure. Cases of hypertrophic laryngitis are slow and troublesome enough, but once well do not recur like a cicatrix. Case XII, one of hypertrophy, had to wear a dilating tube from February, 1904, until March, 1906, and then, owing to adductor spasm, a small tube until the following June, and thus required a little over two years of treatment, which seems to be about the average.

Nothing has been said about the treatment of obturating or compressing neoplasms, as these, if not inoperable, always require removal, and a discussion of the surgery of the air passages does not come within the scope of this article. But a few words about the so-called "exuberant" granulations, which are largely a myth, and about the common papillomas are not out of place. Granulations may, indeed, though rarely, produce obstruction by growing around some source of irritation, like the upper or lower end of a tube, or into the fenestrum on the convexity of a cannula, and then must be cut or scraped away and their recurrence prevented by removal of the cause, which generally means enlarging and rounding off the head of the tube or shortening and smoothing its inferior extremity. The cannula should be replaced by a well-fitting tube. Some means, at any rate, can be always found of doing away with the localized pressure upon an ulcer.

Statistics in cases of multiple papillomas seem to favor tracheotomy and a policy of waiting for an indefinite period, but many surgeons favor one or more attempts at radical removal by an open operation as soon as there are evidences of obstruction, and if the excision could be done thoroughly it would probably be more often advocated. The chief difficulty lies in the abundant capillary oozing and the inaccessibility of the upper part of the larynx and deeper portion of the trachea. To reach these regions the hyoid bone must be divided as well as the thyroid and cricoid cartilages and the trachea possibly down to the episternal notch. To allay the irritability and cough and to check the oozing of blood there is nothing better than a 1 per cent. solution of cocaine and the ordinary 1 to 1000 solution of adrenalin, which can be swabbed freely over the exposed mucous membrane. Then, under a good light and wide retraction of the wound, the base of the fungating masses can be trimmed out with curved scissors well into sound tissue. By inserting one finger through the mouth all of the upper parts of the larynx can be pressed down into sight and treated in the same manner, giving particular attention to the natural folds just above the cords. Then at the end of the operation I have been accustomed to insert a full sized special intubation tube somewhat more cylindrical than the ordinary kind, and with a larger head to insure against the possibility of its slipping down into the weakened larynx. Over it the

wound is closed with two tiers of catgut sutures, and drainage by a few strands of the same material is inserted at the upper and the lower angles. The instrument used in this way prevents appreciable subsequent hemorrhage by its pressure, avoids the danger of œdema and stenosis, and hastens a restoration to the normal. In a conconsiderable experience I have never noted any ill effects. The only risk seems to be that of auto-extubation followed by swelling, which should not occur before the tube could be reinserted or the trachea opened. No infection of any moment has occurred in my cases, although those treated in this manner do not include extensive resections of portions of the whole wall of the trachea or larynx, which, of course, opens the deep cellular spaces of the neck. But even then there are many instances when with proper drainage the wound in the respiratory tract can be closed with advantage over a suitable tube.

THE REACTION OF THE PHAGOCYTES OF ELEPHANT'S BLOOD ON THE BACILLI OF TUBERCULOSIS

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In casting about for an animal whose blood might be availed of in conducting experiments on immunity against tuberculosis, it occurred to me that the elephant possessed two characteristics which might make it useful for this purpose: First, its remarkable longevity, suggesting a power of resistance to all infections, whether of human beings or of the lower animals; and second, the fact that no records could be found of the observation of tuberculosis in this race.

With the skilful coöperation of Dr. Herbert Fox, chemist of the Laboratories of the State Department of Health, the following observations were undertaken. By the courteous permission of the Zoological Society of Philadelphia, a small quantity of blood was obtained from an elephant's tail. At the same time experiments were made with the blood of the human being, the cow, and the guinea-pig. The conditions in each case were identical. The leukocytes were twice washed and the emulsion of tubercle bacilli was prepared by washing lightly with alcohol and ether and rubbing up in a mortar in 0.86 per cent. salt solution and then treating with the centrifuge. The turbidity of the emulsion corresponded to No. 2 of a barium sulphate nephelometer (Table I). A second experiment was made in which the emulsion was produced by boiling in chloroform, the object being to get a suspension which will keep indefinitely and may always be rendered homogeneous by shaking.

Four salt solutions of different strengths were used for the purpose of testing spontaneous phagocytosis.

TABLE I.—COMPARATIVE PHAGOCYTOSIS OF HUMAN, BOVINE, AND RODENT BLOOD WITH BACILLUS TUBERCULOSIS.

	Phagocytic Index.	Average.
0.1 c.c. elephant's serum plus 0.1 c.c. elephant's leukocytes plus 2 c.c. bacteria emulsion	1.0	0.40
0.1 c.c. elephant's serum plus 0.1 c.c. guinea-pig's leukocytes plus 2 c.c. bacteria emulsion	0.7	0.28
0.1 c.c. elephant's serum plus 0.1 c.c. emulsion human leukocytes plus 2 c.c. bacteria emulsion	0.4	0.16
0.1 c.c. elephant's serum plus 0.1 c.c. emulsion cow's leukocytes plus 2 c.c. bacteria emulsion	0.3	0.12
0.1 c.c. cow's serum plus 0.1 c.c. emulsion cow's leukocytes plus 2 c.c. bacteria emulsion	1.0	0.28
0.1 c.c. cow's serum plus 0.1 c.c. emulsion human leukocytes plus 2 c.c. bacteria emulsion	0.57	0.16
0.1 c.c. cow's serum plus 0.1 c.c. elephant's leukocytes emulsion plus 2 c.c. bacteria emulsion	0.43	0.12
0.1 c.c. cow's serum plus 0.1 c.c. emulsion guinea-pig's leukocytes plus 2 c.c. bacteria emulsion	0.43	0.12
0.1 c.c. human serum plus 0.1 c.c. human leukocytes plus 2 c.c. bacteria emulsion	1.0	0.24
0.1 c.c human serum plus 0.1 c.c. emulsion cow's leukocytes plus 2 c.c. bacteria emulsion	0.83	0.20
0.1 c.c. human serum plus 0.1 c.c. emulsion elephant's leukocytes plus 2 c.c. bacteria emulsion	0.5	0.12
0.1 c.c. human serum plus 0.1 c.c. guinea-pig's leukocytes plus 2 c.c. bacteria emulsion	0.33	0.08
0.1 c.c. guinea-pig's serum plus 0.1 c.c. guinea-pig's leukocytes plus 2 c.c. bacteria emulsion	1.0	0.28
0.1 c.c. guinea-pig's serum plus 0.1 c.c. emulsion human leukocytes plus 2 c.c. bacteria emulsion	0.85	0.24
0.1 c.c. guinea-pig's serum plus 0.1 c.c. emulsion elephant's leukocytes plus 2 c.c. bacteria emulsion.	0.57	0.16
0.1 c.c. guinea-pig's serum plus 0.1 c.c. cow's leukocytes emulsion plus 2 c.c. bacteria emulsion	0.4	0.12

One half hour at 37° C. 100 cells counted.

TABLE II.—AVERAGE PHAGOCYTOSIS OF HUMAN AND ELEPHANT'S BLOOD WITH BACILLUS TUBERCULOSIS.

Emulsion made in NaCl solution of	Human. March 29, 1907	Human. March 30.	Human. April 1.	Human. April 2.	Elephant. April 2.
0.1 per cent.	0.46	0.42	0.28	0.30	0.36
0.6 per cent.	0.36	0.28	0.30	0.34	0.34
0.86 per cent.	0.28	0.32	0.24	0.32	0.48
1.5 per cent.	0.32	0.40	0.44	0.38	0.46

In both experiments the blood of the same individual was used, the bacteria for the different emulsions were obtained from the same tuberculous strain, and the turbidity was identical. In the second the whole blood method was employed, using homologous cells and serum. Table II shows the phagocytic capacity of the blood of the same person on four successive days, the last time on that of the second elephant test.

The results of these experiments may be summed up as follows:

1. The expectation that the leukocytes of elephants' blood might possess a high degree of phagocytic energy as far as the tubercle bacilli is concerned was not fulfilled to such a degree as to promise any practical result, being but a trifle higher than that of human cells.

2. The highest phagocytic average is obtained when the leukocytes and cells of the same animal or race are employed.

3. When the serum of the various animals is mixed with the leukocytes of other animals, the results vary very much, in accordance with the order in which the mixture is made. For example, when elephant serum is added to guinea-pig leukocytes, the index resulting is only 0.7, but if the guinea-pig serum is added to elephant's leukocytes, the index mounts to 0.85. If bovine serum is added to human leukocytes, we obtain an index of 0.57, but if human serum is mixed with bovine leukocytes, it reaches 0.83.

4. It seems to make very little difference numerically in the phagocytosis of tubercle bacilli whether they are emulsified by rubbing in a mortar with salt solution or by boiling in chloroform.

REVIEWS.

THE OPERATIONS OF SURGERY. By W. H. A. JACOBSON and R. P. ROWLANDS. Fifth edition; 2 volumes; pp. 926, 1139; 776 illustrations. Philadelphia: P. Blakiston's Son & Co., 1908.

WE have never understood why Mr. Jacobson should insist—as he has always insisted—that his admirable treatise on operative surgery is "intended especially for the use of those recently appointed on a hospital staff and for those preparing for the higher examinations," and "*not* for those with a large surgical experience." There has been no time, during the two decades that have elapsed since its first appearance, when it was not potentially useful to any surgeon, however large his experience, who realized that he had yet something to learn. No other book on the same subject discussed so thoroughly the facts bearing upon what is, after all, fundamental for operators, the decision whether to operate or not, or has given, to aid them in any particular instance, so many illustrative cases from both the personal experience of the author and surgical literature. Very few have excelled it in the recital of operative details. It remains now, in 1908, as it began, in 1888, one of the most satisfactory books that a surgeon can turn to if he desires a broad and comprehensive view of an operative procedure, including some consideration of the precedent and attendant circumstances and the probable result. No other foreign book has, during the same twenty years, so continuously demonstrated entire familiarity on the part of its author with the surgical work that was being done in this country, or has so consistently set forth the results of that work, as have the successive editions of Mr. Jacobson's *Operations of Surgery*.

It has been so frequently and so thoroughly reviewed as to its general teachings, that the chief interest attaching to this edition lies in the present views of the authors as to operations or methods introduced, or popularized, or more widely adopted and extended, or distinctly modified during the last four years, that is, since the appearance of the last previous edition.

Reading it with this idea in mind, the following matters—taken almost at haphazard, to serve as examples—seem worthy of note:

Tendon transplantation is carefully reviewed, the favorable opinions of Tubby and Jones are contrasted with the less favorable

ones of Keetley and Clarke, and the remark of Montgomery (as to the operation in cases of infantile paralysis), "The more complex, the more indirect, the more ingenious the method, the worse the result; the directer, the simpler, the more obvious, the better," is quoted with approval. The authors add: "From America, where every fresh operation is at once tested largely and with much zest, we have warnings not to expect too much from tendon transplantation in infantile paralysis."

In amputation at the elbow joint the possible advantages of Miller's modification of the circular method (heretofore thought to be inapplicable because the cicatrix is apt to be central and adherent) are fully set forth.

As to the treatment of combined shock and collapse, a most useful comparison of the various methods of infusion and transfusion (founded on the recent work of Beddard) has been added, and their relative advantages and disadvantages are clearly stated; and later, the prevention of shock by the preliminary cocainization of large nerve trunks (Crile and Cushing) is adequately discussed. The work of Barrows and Bauer in the intravenous injection of antiseptics in sepsis is also summarized.

In noting the fact that excision of the shoulder joint is rarely performed because of the comparative infrequency of diseases of that joint, especially of tuberculous disease, which require operative measures, the further explanation might have been added that the diseases produced by trauma to the shoulder are relatively infrequent in this joint because of its ample covering of muscles which protect it from effects of cold and damp; the mobility of the scapular segment of the shoulder girdle lessens greatly the effect of traumatisms; the laxity of its capsular and synovial elements, which, though it favors luxation, permits a moderate effusion to occur without harmful tension; the influence of the weight of the upper extremity in the usual position of the body in resisting, by gravity, the destructive pressure of joint surfaces against each other, caused by muscular spasm after injury or during disease; and the ease with which the joint may be immobilized without irksome confinement of the patient (Piersol).

As to the light treatment of lupus, while recognizing fully the vast improvement it brings about in both the ulcers and the co-existent scars, the authors think that we are now only beginning to be in a position to estimate its real "curative" value. They think that many cases of "cures" are valueless because they have been imperfectly or prematurely reported, and remind their readers of the well-known dictum of Verneuil, that operations on the tuberculous abound in "half successes, incomplete results, and unfinished cures."

The subcutaneous injection of sterilized paraffin for the correction of deformities is described, and Mr. Stephen Paget's excellent results are alluded to, but the extravagance of some of the claims,

in reference to the method, and the possibility of late suppuration, are properly mentioned.

Kocher's abandonment of the submaxillary operation for removal of the tongue and adoption of "Syme's operation" (with division of the symphysis menti) are discussed, with approval as applied to cases in which a growth has extended toward the hyoid. In other cases, Mr. Jacobson's original preference for the Whitehead operation remains unchanged.

As to the important question of the period of freedom from recurrence, after the removal of a cancerous breast, which justifies the statement that the patient is "cured," he does not mince matters, but says: "I look upon Volkmann's teaching as most pernicious, and on the adoption of his teaching—that if patients remain free for three years after an operation for malignant disease, they may be looked upon as cured—as alike unscientific and dishonest, and unworthy of our profession." Most surgeons who are more occupied with observing facts than with making statistics will agree with him. He rightly thinks that when the supraclavicular glands are involved in such cases the condition is hopeless so far as radical cure is concerned, and believes it is analogous to that found in cancer of the tongue, when the glands of the neck are involved, quoting Mikulicz in support of this opinion. The whole article on the removal of the breast is one of the soundest and most instructive in the book.

For the radical cure of femoral hernia, he thinks Lotheissen's operation (the essential part of which is the closure of the *upper end* of the femoral canal by joining the lower margins of the internal oblique and transversalis to Cooper's ligament) is the most anatomically ideal, and is, therefore, destined to become the operation of the future.

The surgical treatment of Bright's disease by Harrison's method (renipuncture, capsulotomy, or nephrotomy), or by Edebohls' "renal decapsulation," is considered from both the theoretical and the statistical standpoint. He concludes that: "Published facts do not lead us to hope for favorable results, from surgical interference in Bright's disease," and that "experimental evidence lends but little if any support to the advocates of this operation, and it fails to demonstrate any real anatomical reason for its supposed success. The literature of the subject is full of vagueness, inaccuracy, and confusion."

The sections on operations on the ureter, retroperitoneal hernia, appendicitis, gastrojejunostomy, castration and vasectomy, prostatectomy, prolapsus ani, excision of the rectum, aneurysm, ectopia vesicæ and epispadias, and many others, have been recast or so largely added to as to make them almost new articles; they tempt to quotation, but space does not permit.

In every one of those mentioned, and, indeed, throughout the book, the recent writings and methods of American surgeons are not merely fully described, but often constitute the chief material that has been added; and many of the illustrative cases and no inconsiderable number of the cuts are abstracted or redrawn (always with full credit) from the pages of American journals.

This edition is at once evidence of the continued industry, scholarship, and sound judgment of its authors, and a tribute to the value of the surgical thought and accomplishment of our country at the present day. J. W. W.

A System of Medicine. By Many Writers. Edited by Thomas Clifford Allbutt, M.D., LL.D., F.R.C.P., F.R.S., Regius Professor of Physic in the University of Cambridge; and Humphry Davy Rolleston, M.D., F.R.C.P., Physician to St. George's Hospital, London. Vol. II, Part I, pp. 1087; Part II, pp. 1055. London: Macmillan & Co.; New York: The Macmillan Company, 1906.

The original Vol II of Allbutt's *System of Medicine* is issued in the revised edition in two parts, both of which, however, are as large as the original single volume. Part I is devoted to sundry infectious diseases, and Part II to tropical diseases and animal parasitic infections. The opening article is an exhaustive account (198 pages), by James Ritchie, of the general pathology of infection—in part a revision of an earlier article by the late Professor Kanthack, but in greater part entirely new. It is one of the most complete and satisfying articles on the subject extant—a credit alike to the author and the *System* of which it forms a part. Then follows a discussion of certain infective diseases of established bacteriology: Glanders and farcy, by G. Sims Woodhead; anthrax, by John H. Bell and Thomas M. Legge; tuberculosis, by Sidney Martin and W. C. Bosanquet—a valuable summary of the general pathology, modes of infection, spread of the disease in the body after infection, sources of infection, immunity, etc.; the pathology of streptothrix infections, by A. G. R. Foulerton, comprising among other important matters a discussion of the relationship of Bacillus tuberculosis to the acid-fast streptothrichæ, to which botanical group the author is inclined to believe Bacillus tuberculosis belongs; actinomycosis, by T. D. Acland; and syphilis, by Jonathan Hutchinson.

An excellent section on infective diseases of doubtful nature is contributed as follows: Measles, German measles, infectious coryza, and glandular fever, by Dawson Williams; scarlet fever, by F. Foord Caiger and L. S. Dudgeon; chicken-pox and smallpox, by John MacCombie; typhus fever, by Sir John Moore; whooping-cough

and mumps, by Eustace Smith; rheumatic fever, by Sir William Church and W. Bulloch; and acute rheumatism in childhood, by W. B. Cheadle. All of the articles are fully abreast of current knowledge and help materially to maintain the reputations of the respective authors. Thus, Dr. Caiger believes that the discharges from the mouth, nose, and ear in scarlet fever are much more infectious than the desquamating epithelium; Dawson, in a new article on infectious coryza, discusses the bacteriology of a common cold and although he refers to Micrococcus catarrhalis, he is inclined to believe that much the same symptoms may be provoked by different organisms; W. Bulloch discusses the supposed relationship of certain microorganisms to rheumatic fever, but though inclined to the general belief that the disease is an infection, he states that the specific bacterial factor is not known. Vaccinia in man is discussed by T. D. Acland—a contribution worthy of the author and food for reflection by our antivaccination friends; the pathology of vaccinia, by Monckton Copeman; vaccination as a branch of preventive medicine, by John C. McVail; foot-and-mouth disease, by Sir John McFadyean; hydrophobia, by G. Sims Woodhead; and the co-exisence of infectious disease, by F. Foord Caiger.

The final section of the book is devoted to intoxications: Food poisoning, by H. Batty Shaw; grain poisoning, and opium and other intoxicants, by T. Clifford Allbutt and W. E. Dixon; alcoholism, by H. D. Rolleston; and metallic and other poisons, including poisonous trades, by T. Oliver. Dr. Shaw makes the interesting and important suggestion that ptomaine poisoning as such is a very restricted possibility, and that the symptoms following the ingestion of tainted food are due to pathogenetic organisms that were present before death in the animal from whch the food was obtained, or that have gained access to the food subsequently. The articles by Professors Allbutt and Dixon are such that most of us will want to read them.

Part II of Volume II, as now issued, is a complete treatise on tropical diseases. Some of the articles were contributed to the former edition; these have been revised and removed from their former position, but the majority are new. In order to provide an authoritative account of the animal parasites and carriers of tropical diseases, special articles by zoölogists have been included: Protozoa, by E. A. Minchin; mosquitoes by F. V. Theobald; blood-sucking and other flies known or likely to be concerned in the spread of disease, by Ernest E. Austin; and ticks, by R. I. Pocock. These, comprising over 200 pages, form an exceedingly valuable addition to the book. The long list of tropical diseases is admirably treated by acknowledged authorities. The final section of the volume is devoted to an excellent discussion of animal parasitic infections.

The two volumes form a noteworthy addition to contemporary medical literature; they are almost indispensable to physicians who would keep abreast of the progress of medical science. A. K.

HANDBOOK OF CUTANEOUS THERAPEUTICS. By W. A. HARDAWAY, M.D., LL.D., Professor of Diseases of the Skin and Syphilis in the Washington University, St. Louis, and JOSEPH GRINDON, Ph.B., M.D., Professor of Clinical Dermatology and Syphilis in the Washington University, St. Louis. Pp. 606. Philadelphia and New York: Lea Brothers & Co., 1907.

IT may not be known to every reader that Professor Hardaway published a *Manual of Skin Diseases* some years ago, which was an eminently useful and practical little work replete with originality. We are gratified, therefore, to learn that in association with Professor Grindon, also favorably known as an author, he has conjointly given his pen to the book before us, just published. It is built upon lines that are eminently practical, brief descriptions of the diseases viewed from the clinical standpoint, followed by sufficiently extended remarks upon the treatment, together with abundant formulas, being given. It is intended for the general physician and not for the specialist. Common-sense views and broad ideas, clothed in language that is straight and unequivocal, characterize the substance and style. There is no ambiguity, no disposition to straddle the question and thus occupy non-committal or neutral ground, as authors too often are wont to do. Doubtful questions and problems are frankly met and courageously disposed of, sometimes perhaps rather too briefly, but the volume, it is to be remembered, is a handbook and not a treatise. The nomenclature is sound, with a distinctive tendency to the leading American and English lines of thought. The classification of the diseases is also considered in the same vein. The formulas (of which there are a large number) are written in Latin. We are pleased to note that the English weights and measures are strictly adhered to throughout the pages, which for American and English readers renders the formulas readily workable and easy to to remember. Some excellent recent books have been overweighted with the combination of the English and the Continental weights and measures, to be accounted for possibly by a desire on the part of the author to have his book considered "advanced."

A relatively lengthy chapter is devoted to general treatment and methods, and here may be observed the large experience and conservatism of the authors, as well as the enunciation in a concise practical form of the application of the principles of therapeutics. This is much the most interesting and valuable chapter in the book. The remarks on the use of the Röntgen-rays are sufficiently full, and are conservative, as they should be. The very few words devoted to radium would indicate extra conservatism. More, we think, might safely have been said as to this therapeutic agent and its value in selected cases. Occasionally in formulas one notes the employment of a proprietary remedy, which is to be regretted; the less the student and practitioner hear of secret formulas the better.

The authors of all countries are drawn upon and liberally quoted, but the references are seldom given, doubtless for want of space. As already observed, the tenor of the train of thought throughout is distinctly and almost exclusively clinical, the subjects of etiology, pathology, and pathological anatomy being only lightly or not at all touched upon. The lack of information of this kind is in some instances noticeable. Thus, in considering impetigo contagiosa, the presence in the lesions of the streptococcus is not mentioned. Reference, moreover, might also have been made to the fact that the lesions in this disease are always extremely superficial, as shown by sections of the skin under the microscope. It would add to the value of the book if this aspect of the diseases in general were entered into more fully in future editions.

We gladly welcome books of this kind, written by authors who do credit not only to the specialty itself, but also to general medicine. In fact, the period has arrived when all the broad specialties in their general pathology and treatment cannot be regarded and treated otherwise than as a part of general medicine. A cordial reception should be accorded this useful handbook, well adapted to the needs of the student and the general practitioner. L. A. D.

EPILEPSY: A STUDY OF THE IDIOPATHIC DISEASE. By WILLIAM ALDREN TURNER, M.D. Edin. Pp. 272. New York and London: Macmillan & Co., 1907.

No one has a better right to discuss epilepsy than Dr. Turner, whose work upon this subject has been so well known for many years. His observations are based upon the study of over 1000 cases. He considers the greatest predisposing cause to be a neuropathic predisposition which manifests itself in various psychical, pathological, and structural peculiarities. He obtained such a history in about 50 per cent. of his cases, but its absence, in his opinion, does not imply its non-existence, in view of the presence of well-marked stigmata of degeneration in many of the cases. He considers that the most common predisposing cause is ancestral epilepsy and that insanity and parental alcoholism exert only a minor and secondary influence.

He states that the most common demonstrable cause of epilepsy is infantile convulsions. Out of 1000 cases 10 per cent. commenced during the first year of life and 34 cases, or 8.7 per cent., had convulsions during infancy and subsequently developed epileptic fits.

The eclampsia associated with the onset of infantile hemiplegia was in 78 per cent. of the cases followed by epilepsy in later life, and he considers that the convulsions associated with the exanthemata, acute infective disorders, and disorders due to toxic influence, including some forms of puerperal eclampsia, should be regarded

as manifestations of acute epilepsy. Dr. Turner classifies epileptic attacks into, first, minor epilepsy, dividing this into incomplete and complete attacks; second, into major epilepsy with similar divisions; and, third, psychic epilepsy. In his opinion the feature that is necessary to establish the existence of epilepsy is sudden temporary loss or impairment of consciousness.

When discussing the mental states he classifies these into, first, the epileptic character and temperament; second, the paroxysmal psychoses which either precede or succeed the convulsive phenomena; third, the paroxysmal psychoses which replace single fits or a series of fits, and which have been regarded as psychic equivalents of the convulsive seizures; and, fourth, the permanent interparoxysmal mental condition which is observed in most epileptics, and which should be looked upon as a well-marked and characteristic feature of the confirmed disease. This classification is about as satisfactory as can be made.

The medicolegal importance of the mental states which occur in this disease, especially in the type known as psychic epilepsy, are of the greatest importance. A number of cases are on record in which patients supposed to be in the condition of psychic epilepsy have committed homicide and in the subsequent trials this defence was made. It is rather important to know whether it is possible for a patient who has psychic epilepsy to recall some or all of the details after his attack. The preponderance of evidence seems to be that this cannot be.

The chapter on the pathology of epilepsy was written by Dr. John Turner. He believes that epilepsy is a disease occurring in persons with a defectively developed nervous system, indicated by certain structural peculiarities, and in whom there is a special tendency to intravascular clotting; that the convulsions are a symptom of the disease; that prior to a seizure cortical stasis is induced by the formation of intravascular coagula, which become lodged in some of the cortical bloodvessels; and that in the process of coagulation the blood plates play a prominent part. It is also contended that intravascular coagulation will explain the atrophy and the sclerosis, and the blood tumors found in many epileptic brains. In the former the blockage is believed to take place on the arterial side, leading to starvation of the cortical tissues; while the latter is explained by blocking of the veins so as to cause a local rise in arterial pressure, which results in enormous dilatation of the capillaries.

Dr. Turner defines the cure of epilepsy as the arrest of the seizures for a period of eight or nine years, and according to him it will be found that from 10 to 12 per cent. of cures may be expected. This is a very common-sense view and one to which most neurologists will subscribe. Altogether this is an excellent and, without doubt, the best presentation of the subject of epilepsy. T. H. W.

A MANUAL OF HYGIENE AND SANITATION. By SENECA EGBERT, A.M., M.D., Professor of Hygiene and Dean of the Medico-Chirurgical College of Philadelphia. Fourth edition; pp. 498; 93 engravings. Philadelphia and New York: Lea Brothers & Co., 1907.

THE feature of this book which first impressed the reviewer is the judicious economizing of space which has made it possible to cover the various subdivisions of the subject in an adequate, if not exhaustive, manner within the limits of so small a number of octavo pages as 498, and still leave room for illustrations. Bacteriology is disposed of in one chapter, part of which is devoted to a summary of the theories of immunity. The chapters on ventilation and heating and on the disposal of sewage and house plumbing deal with matters that interest every citizen and householder. The information on school hygiene, both within the schoolhouse and as it applies to the children's home life, and the discussion of practical school questions, such as the proper system of heating and ventilating the schoolroom, the most approved kind of desk and its disposition with reference to the light, the placing of blackboards, the child's position while sitting at the desk, the care of the eyes, and overworking of school children, should be of great value to those intrusted with the care of children and especially to our medical school inspectors. Military hygiene deals briefly with "all phases of the routine life of the soldier," from the selection of recruits to the prevention or control of typhoid fever in camps and barracks. In this chapter the author quotes freely from Vaughn's oration on State medicine, delivered before the annual meeting of the American Medical Association in 1900, and contrasts the conditions which obtained in the United States army in 1898 with the brilliant sanitary record of the Japanese in the Russo-Japanese war, which he attributes to the greater authority enjoyed by the Japanese medical officers in all matters pertaining to sanitation. More thorough instruction in the fundamental principles of hygiene is urged for the national military training school. In the closing chapter the author gives a number of simple methods for the examination of samples of air, water, and foodstuffs, that are within the reach of anyone possessing ordinary scientific training, and such simple apparatus as can be easily obtained or improvised without much expense. The subjects so treated include, besides air and water, milk, butter and oleomargarine, flour, and bread. The author's manner of presentation is clear and concise, his style is as simple as the subject permits, and the book is equally well adapted to the needs of the layman in search of information on practical matters and to those of the municipal or military medical officer who desires a convenient reference handbook.

R. M. G.

A Manual of Surgery for Students and Surgeons. By Francis T. Stewart, M.D., Professor of Surgery in the Philadelphia Polyclinic and College for Graduates in Medicine. Pp. 778; 504 illustrations. Philadelphia: P. Blakiston's Son & Co., 1907.

This book was "prepared for the undergraduate whose crowded hours demand a manual stripped of verbiage and unessentials, and for the medical practitioner who seeks a guide to present day surgery." There seems to be a demand by undergraduate students for books of this class, and if such must be supplied the author is to be congratulated for producing a volume of such general excellence. It is a welcome escape from the abominable little quiz compends which flood the medical book market. But this should not be construed to apply to the general practitioner who requires in his every day practice some means of acquiring knowledge of the essentials and of the minutiæ of technique which is usually omitted. The volume, which includes genito-urinary diseases, gynecology, and the essentials of minor surgery, is modern in most particulars, and is much to be commended. G. P. M.

La Pratique de la Médecine. Par William Osler, Professeur royal a l'Université d'Oxford. Traduction française sur la 6e edition par M. Solomon, Chef de clinique a la Faculte, et Louis Lazard, Ancien interne de l'Hôpital de Rothschild. Preface du Dr. Pierre Marie. Paris: G. Steinheil, 1908.

It is a source of much pleasure to chronicle the publication of a French edition of Osler's *Practice of Medicine*. Since the book has gone through six large editions in English and is well and favorably known throughout the English-speaking world, little need be said of its intrinsic excellencies. Special interest in the present connection centres in the translation—which has been admirably done, preserving much of the personal flavor, terseness, and lucidity of the original. We commend the book to our French confrères.

A. K.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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Metallic Resonance.—R. GEIGEL (*Deut. Arch. klin. Med.*, 1907, xc) states that although metallic resonance is an important phenomenon in physical diagnosis, and undoubtedly forms a most valuable sign in diagnosticating air-containing cavities, yet there has hitherto been no satisfactory explanation for the phenomenon. A. Geigel (1856) suggested the higher overtones as a cause, but he did not explain the mechanical conditions for their production. There must first be a clear conception of what is meant by metallic resonance. Physics recognizes simple and compound tones, the latter consisting of fundamental tones and overtones. The lower overtones fuse with the fundamental tone, the higher ones do not, and these produce the phenomenon of metallic resonance. Helmholtz discussed fully the conditions and theory of the production of the higher overtones, and it it is only necessary to repeat briefly his conclusions and to show that the conditions supposed by him actually exist whenever in physical diagnosis we meet with metallic resonance. In any vibrating body the production of the fundamental tone is favored by the continuity of the velocity of the different parts of that body. If there are abrupt transitions in the velocity of neighboring particles, the resultant movement is termed discontinuous, and favors the production of higher overtones with suppression of the fundamental tone. Factors in the production of discontinuous vibration are a hard hammer, a rapid stroke, and a tense, vibrating body, since these hinder the rapid distribution of motion to all parts of the vibrating body.

We may now apply these principles to percussion and auscultation. In eliciting metallic resonance over a cavity, the stroke should be short and sharp and best made with a hammer and pleximeter. The wall of the cavity must be tense. The cavity must be near the chest wall, else

the intervening tissue will distribute the impulse over too large a part of the cavity wall, and thus hinder the production of discontinuous motion in the contained air. The cavity must not be too small, else the overtones are too high pitched to be perceived by the ear. Inflated bladders yield at first only a tympanitic note on percussion; with greater inflation, a metallic, note since great tension of the wall is required for the production of discontinuous movements in the contained air. That the note is produced by the air and not by the bladder wall is shown by the greater depth of the tone as the bladder is inflated and the volume of air increased. A higher pitched note would be expected were it produced by the wall itself. Instead, there is a deeper note with the addition of metallic resonance.

In order that the blast of air across the mouth of a jar or bottle should elicit metallic resonance the mouth must be relatively narrow as compared with the greatest diameter of the bottle, and the transition between the two diameters should be abrupt. Discontinuous movements in the contained air may then be produced. The same holds for the production of amphoric breathing in cavities. Other factors are a rapid current and conditions in the bronchi which themselves give rise to discontinuous blasts. Tense, reflecting walls will not in themselves produce amphoric breathing. Falling drops by suddenly disturbing the equilibrium of the contained air at one point produce the characteristic tinkling sound. Metallic rales owe their origin to a discontinuous vibration imparted to the air by the sudden rupture of tough particles. In succussion the metallic sound is produced by the effect upon the air of sudden changes in the surface of the contained fluid. Such changes are impossible in cavities containing viscid secretions. Sometimes metallic sounds are auscultated when the cavities are completely shut off from the air current. In these instances the sounds originate elsewhere and the metallic resonance is a secondary element added by the cavity, which we may think of as having been subjected to a kind of percussion. Examples are amphoric breathing in the neighborhood of a cavity not opening into any bronchus and enclosed pneumothorax; and metallic heart sounds in pneumothorax and in extreme gastric dilatation.

A Granular Form of Tubercle Bacillus Unstainable by Ziehl's Method.—Much (*Beit. z. klin. Tub.*, 1907, viii, 85) comments how frequently one is unable to demonstrate acid-fast bacilli in smears from pearl nodules of cattle when inoculation and culture show the tuberculous nature of the lesion. The same is true of cold abscesses in man. Clinicians have noted, too, the sudden disappearance of tubercle bacilli from sputum in which they had previously been abundant, and after an interval their sudden reappearance. In stained smears from tuberculous lesions in guinea-pigs Much found that frequently a much larger number of organisms could be demonstrated by means of Gram's stain than by the Ziehl or Ehrlich method. Frequently, too, granules were seen taking the same color tone as the bacilli. These granules may be arranged in a typical rod shape, or may be heaped in masses bearing no resemblance to the bacilli. It is these glanular masses which are not stainable by the Ziehl method that Much looks upon as an unusual form of the tubercle bacillus.

A calf that had been inoculated with $\frac{1}{20}$ mg. of virulent cattle tubercle bacilli was sectioned and its lungs found studded with gray miliary tubercles. A large number of smears were stained by Ziehl's method and carefully searched, but no tubercle bacilli were found. Gram's method also gave negative results. Sections of the lung were then cut and stained, and over fifty preparations examined without finding tuberlc bacilli or any acid-fast bodies. The usual Gram method was likewise negative, but after leaving the sections for forty-eight hours in aniline gentian violet, and immersing for from five to ten minutes in Lugol's solution, a very unexpected and striking picture was obtained. The tubercles were found filled with small slender rods and in places with granules arranged as rods or heaped in irregular piles. Cultures from the tubercles showed typical tubercle bacilli after fourteen days in the thermostat. A tube removed each day and examined showed by Gram's stain slender rods and granules on the third day, but no acid-fast organisms until the sixth. Pieces of the lung tissue injected into guinea-pigs caused death from general tuberculosis. Three sheep injected with the same culture showed general pulmonary tuberculosis.

In the pus from cold abscesses which contained no bacilli demonstrable by the Ziehl method, but which produced tuberculosis in animals with numerous acid-fast bacilli demonstrable in the lesions, Much found the Gram-staining granules. He was able to show that tubercle bacilli inoculated into milk rapidly lost their acid-fast quality, but were long afterward demonstrable by Gram's method, first as slender rods, then as heaps of granules. As long as these granules were present the milk retained its virulence for guinea-pigs. Much summarizes his work in the following conclusions: (1) There is a granular form of the tubercle bacillus which cannot be demonstrated by Ziehl's method; (2) this granular form is virulent; (3) it may occur in tuberculous organs as the only stainable manifestation of the tubercle-producing agent; (4) it may be associated with a rod form likewise unstainable by Ziehl's method; and (5) there are transition stages, from the granules stainable only by Gram to the Gram-staining rods and to the ordinary acid-fast bacilli. Much suggests that carbol-fuchsin stains a different substance in the tubercle bacillus from the gentian violet. Its acid-fast properties probably depend upon the fats it contains.

Skin and Ophthalmic Reactions to Tuberculin.—MAININI (*Münch. med. Woch.*, 1907, liv, 2583) refers to the fact that von Pirquet has found that by applying dilute solutions of old tuberculin to an area of scarified skin a red, erythematous papule develops in individuals with tuberculosis, while no reaction occurs in the non-tuberculous. An extended use of the method has shown that so many apparently healthy adults react that it is useless in diagnosis except in very young children. This large number of reactions corresponds to the frequency with which unsuspected tuberculosis is found at autopsy. In 360 children von Pirquet found that 88 per cent. of the tuberculous reacted positively, the negative cases being advanced or miliary tuberculosis. In 109 non-tuberculous nurslings, only 2 reacted positively. In children from eight to fourteen years of age, however, 35 per cent. of unsuspected cases

gave a reaction. In 100 autopsies on children from one to fourteen years of age, who had been vaccinated, he found 30 cases that had reacted all tuberculous, and 17 more of miliary or very advanced tuberculosis that had not reacted. The other 54 were free from tuberculosis and only one had reacted. Mainini reports his observations on 208 cases. Of 24 definitely tuberculous cases, 19 were positive. The 4 negative had very advanced tuberculosis. Of 74 suspected cases, 67 reacted. Of the 7 negative cases, 2 died shortly afterward of advanced tuberculosis. Of 111 patients suffering from various diseases, but in whom there was no cause for suspecting a tuberculous lesion, 89 reacted.

Shortly after the publication of von Pirquet's method, Wolf-Eisner proposed the instillation of tuberculin into the conjunctiva, tuberculous individuals reacting by a reddening of the sac or more marked inflammation. Calmette modified this reaction by using a purified tuberculin. Mainini, in his observations, has used a 5 per cent. solution of the old tuberculin. Of 12 cases of tuberculosis, 11 reacted, the 1 negative being an advanced cae. Of 32 suspected cases, 26 reacted. Of 56 non-tuberculous cases, 8 reacted. These same 100 cases showed the following relation to the skin reaction: In Group 1, 11 cases were positive; in Group 2, 31 were positive; in Group 3, 50 were positive. It is seen that the greatest discrepancy is in the non-tuberculous group, and if the reaction is specific this indicates a greater sensitiveness on the part of the skin. Mainini presumes that more of the specific substances are centred in the skin than in the conjunctiva, which, therefore, requires more tuberculin to liberate a reaction. As is well known, one dose of tuberculin sensitizes to a second, and he suggests the possibility of producing a local hypersensitivesness by repeated instillations. Of 19 cases which failed to react to the first dose, 15 reacted to a second in the same eye. Apparently such repeated applications would put the eye reaction on the same percentage level as the skin reaction. Mainini's conclusions are that the cutaneous as well as the ophthalmic reaction gives a constantly positive result in cases of definite tuberculosis except in those that are far advanced. The specificity of the reaction is probable, but as yet not definitely proved. In non-tuberculous individuals the cutaneous reaction gives about six times higher percentages than the ophthalmic reaction. Assuming that the reaction is specific, this apparently contradictory result leads him to conclude that the ophthalmic reaction indicates more accurately active tuberculous lesions, while the skin reaction includes latent foci.

Influence of the Antiferment of the Blood on the Proteolytic Leukocyte Ferment.—Wiens (*Deut. Arch. f. klin. Med.*, 1907, lxxxi, 456). Müller and Jochmann have shown that washed leukocytes contain a ferment that digests proteids, but that this ferment action is lost if even a small amount of blood plasma be added. Müller and Kolaczek found a marked proteolytic action exerted by fresh pus diluted 640 times, this effect being inhibited by the addition of from five to ten times this amount of serum. This inhibitory action of the serum varies in extent in different individuals and under different conditions; Wiens' object was to determine if such variation is constant and what relation it bears to other pathological phenomena and to associated blood changes. In infectious diseases there is a wide range of variation, but a general

principle is found to obtain, namely, that diseases associated with a leukocytosis have a low inhibitive power serum. This is due to the large amount of antiferment used by the disintegrating leukocytes. In 3 cases of sepsis, one a rapidly fatal streptococcic infection, had a high antiferment index; 2 milder cases, a low. Perhaps repeated observations would be of some prognostic value. In cases of pulmonary tuberculosis the index is high, except when a secondary infection associated with a leukocytosis is present. This increase is probably due to the passive congestion. In pneumonia there is usually a marked decrease during the disease with a sudden rise immediately after the crisis, and then a gradual return to the normal. In cases of cardiac disease there is no noticeable variation from the normal. In acute endocarditis the conditions parallel those of sepsis, and in one instance a gradual amelioration of all the symptoms was associated with a definite decrease in the amount of antiferment. Two cases of gastric carcinoma showed no abnormal conditions, while in cases of jaundice there is a decrease. As normal bile contains no antiferment, it must have been altered by the inflammatory changes. In intestinal diseases the quantity of inhibiting substance varies with the extent and character of the lesion. In infections associated with the destruction of leukocyes there is a correspondingly low value. In tuberculous cases it is increased or normal. In only 2 nephritic cases were abnormal conditions found, and in both of these there were abundant pus cells in the urine, indicating an increased destruction of leukocytes. In 3 cases of diabetes and 1 of Basedow's disease there was a low amount of antiferment, for which Wiens could not suggest a satisfactory explanation. The only striking results of the investigation are the lowering of the inhibiting action of the serum in sepsis and its increase in tuberculosis.

SURGERY.

UNDER THE CHARGE OF

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The Value of Blood Pressure Determination in the Diagnosis of Aneurysm of the Thoracic Aorta.—WILLIAMSON (*Lancet*, 1907, ii, 1516) made investigations in 30 cases of aneurysm of the aortic arch, or ascending aorta in which the innominate artery was involved, to determine whether instrumental examination would show a difference in arterial blood pressure between the two upper extremities. He examined the

same point in other series: (1) Cases in which there were the clinical signs of mere dilatation of the ascending part or arch of the aorta; (2) cases either of arteriosclerosis without signs of dilatation of the aorta or else of mediastinal tumors; and (3) cases in which there were no signs of any of these clinical conditions. Stanton's blood-pressure instrument, and Oliver's hemodynomometer were employed. Stanton's instrument gave the more reliable results. Digital examination of the pulses in cases of aortic aneurysm is a far less sensitive method than instrumental, frequently giving results directly at variance with those obtained by the latter means, and hence is of much less value in diagnosis.

Treatment of Urethritis by the Ionization of Silver.—MELUN (*Ann. des maladies des organes génito-urinaires*, 1907, xxv, 1681) says that his method, ionization of silver, especially in chronic urethritis, is as follows: A specially constructed straight, black, hardened, hollow, rubber sound, 25 cm. long, No. 18, is employed. From the tip nearly to the opposite end of the sound are a number of small perforations passing from the external surface to the interior. In the lumen is a silver wire extending almost the entire length of the instrument and passing out through an opening near that end of the sound which is to remain external to the urethra. There the wire is to be placed in contact with the pole of a galvanic battery. The external end of the instrument is fitted with a stop-cock. It is introduced into the urethra until the perforated portion is covered. The meatus is pressed about the instrument by an assistant or the patient himself. The stop-cock is opened and a syringe which fits into the open end of the sound is employed to inject into its cavity about 10 grams of a 1 to 2 per cent. solution of silver nitrate. This solution escapes from the sound by the perforations at the sides into the urethral canal, and produces a distention of the mucosa. This puts the silver wire in the sound in conductible communication with the mucosa. The stop-cock is closed and all is ready for the circulation of the galvanic current. The external end of silver wire is then attached to the positive pole of the battery. The negative pole may be placed at any point on the body of the patient, since it plays an indifferent role. The current is then turned on lightly and may be increased to 30 or 40 milliamperes without provoking shock or pain. The patient tolerates this treatment very well for fifteen to thirty minutes. The silver nitrate is decomposed and the ions penetrate the tissues, glands, and mucosa. This result cannot be obtained by any other treatment. An energetic curative action was thus produced in three cases of chronic urethritis.

Sigmoiditis and Perisigmoiditis; Simple Inflammatory Affections of the Sigmoid Flexure.—PATEL (*Revue de chir.*, 1907, xxvii, 698) says that it will be difficult to detect the presence of an acute, non-suppurative sigmoiditis when a general disease is present. The perisigmoid collection of pus may be an extension from a pelvic infection in the female, a residual phlegmon in the left iliac fossa, a suppurative intraligamentary cyst of the ovary, or a circumscribed focus of peritoneal fibrocaseous tuberculosis. The diagnosis of such a pus collection due to extension from an ulcer of the sigmoid is very difficult. There are no positive

signs, except after operation. Cancer, here, may first be recognized as an abscess. Experience has shown that the evolution of a chronic perisigmoiditis secondary to a sigmoiditis is particularly benign. The infectious process seems to be limited. At times it gives rise to subacute attacks, but it rarely ends in perforation or abscess. Fibrous bands develop around the sigmoid, particularly in its fixed portion and then may lead to an occlusion of the bowel.

Bland-Sutton reported a case, a woman aged sixty years, in which he did a resection for what he thought was an iliac new-growth. The sigmoid flexure was dilated and had rigid walls. In the piece removed he found an epiploic appendage in the centre of which was a fragment of straw. This had been the source of a chronic inflammation which had extended to the loin. The patient recovered without incident. An analagous case was reported by Tuffier, who found some epiploic fringes simply indurated, without thickening of the intestine. The patient got well after a simple laparotomy. Carlson and others believe that these diverticula are more frequently the cause of trouble than is generally thought. Some subacute cases of perisigmoiditis consist of almost acute attacks grafted on an old lesion. They may or may not end in abscess formation. In some cases an iliac swelling of large size may develop with constipation and pain, and the swelling may disappear under the influence of purgatives or proper regimen. Inflammatory strictures may result as the final stage of a sigmoiditis and especially of a perisigmoiditis. Diverticula, when present, do not of themselves cause strictures, but they give rise to a sigmoiditis or perisigmoiditis which determines the stenosis. Two patients of Paviot's died from general peritonitis due to the perforation of a sigmoid inflammatory mass. Inside the bowel in one were some valves forming pouches which contained small fecal masses, and two of these pouches communicated with the abscess and the peritoneal cavity.

Splenectomy for Carcinoma.—SMITH (*Annals of Surgery*, 1908, xlvii, 53) reports a case in which, ten years ago he removed an ovarian cyst, the cavities of which were filled with a gelatinous fluid. The same fluid was also found in the abdominal cavity. The walls of the various compartments were partly smooth and partly covered with a proliferating papillomatous growth. The peritoneum throughout was thickened and vascular, and it as well as the uterus and intestines were profusely studded with a growth resembling wet sago grains. The trouble seemed to begin with a pregnancy three years before. Ten years after the first operation Smith again operated on the patient, this time for a carcinoma of the spleen. The patient recovered. Later the abdomen was opened showing the small nodules of the same semisolid growth in the omentum. The following points of special interest are noted: The ovarian tumors probably developed during or soon after pregnancy. The interval between the removal of the ovarian tumors and the splenic growth was a long one. The sago-like growth disappeared entirely after the first operation, and a similar growth appeared in the spleen. The same colloid growth reappeared in the abdomen after the splenectomy, doubtless taking its origin from a part of the spleen where the growth had burst through the capsule. The

blood showed slight variations, both before and after operation. There were no symptoms pointing to any organ as the cause of the rapid emaciation and loss of strength.

Splenectomy in Banti's Disease.—TORRANCE (*Annals of Surgery*, 1908, xlvii, 41) collected 35 cases from the literature and added a new one of his own. In the first stage of this disease the spleen becomes enlarged without known cause, is firmer than normal but still preserves its normal color. A progressive anemia develops later with occasional periods of remission. The skin becomes bronzed or pigmented with jaundice. This stage may last from three to ten years. In the second stage, which lasts only a few months, the urine becomes scanty, high colored, loaded with urates and contains urobilin. In the third stage there is ascites with Laennec's cirrhosis. As a consequence of vasomotor paresis of the splanchnic area, arising from disease of the visceral sympathetic ganglia, there is great engorgement of the abdominal viscera, especially of the spleen and liver, and increased hemolysis with consequent oligochromemia and oligocythemia. The increased blood supply to these organs leads to fibrosis and lessened function. The peritoneal effusion is due rather to vascularity than to portal obstruction. Of the 36 patients, 9 died, a mortality of 25 per cent.; 17, or 47 per cent., were females; 15, or 42 per cent., were males; in 4, or 11 per cent., the sex was not given; under twenty years of age 5 or 13.9 per cent.; between twenty and forty years, 18 or 50 per cent.; between forty and sixty years, 6 or 16.6 per cent.; age not given in 7, or 19.5 per cent.

Observations on the Treatment of Fractures of the Neck of the Femur.—WALKER (*Annals of Surgery*, 1908, xlvii, 84) says that of 378 fractures of the femur admitted to the Bellevue Hospital, 112 were fractures of the neck; 9 cases occurred in patients under thirty years of age, and 30 cases under fifty years. During the past eighteen months Walker has employed the Whitman method of treatment in 16 cases. This consisted, briefly, in administering an anesthetic and making gentle examination. The patient was then lifted upon a box or pillows about eight inches high and large enough to support the head, shoulders, and trunk. The pelvis rested on a sacral support, and the extended legs were held by assistants. The fractured limb was held at the normal limit of abduction, about 45 degrees, and traction was made at the same time to overcome the shortening. A plaster spica was so applied as to include the pelvis and crossed below the edge of the ribs. It extended to and included the foot, preventing completely any outward rotation. Walker reached the following conclusions: Fractures of the neck of the femur occur more frequently than was formerly believed. Any injury to the hip followed by disability should suggest the possibility of a fracture of the neck, and requires an expert examination aided by an *x*-ray photograph. Reduction of the deformity with complete immobilization of the fracture during the period of repair is advised by means of a plaster bandage in all suitable cases. This is to be followed by early gymnastic movements, active rather than passive. All weight bearing upon the fracture is to be avoided for from three to four months.

Difficulties in the Diagnosis of Abdominal Tumors.—KROEMER (*Mitt. a. d. Grenz. d. Med. u. Chir.*, 1907, xviii, 440) states that the diagnosis of uncomplicated tumors of the female genital organs should offer relatively little difficulty. They do not always remain confined to the pelvis, but may mount into the abdomen, and even completely fill it. In the consideration of differential diagnosis the text-books usually divide these tumors into the small, moderately large, and very large tumors. A small tumor may remain confined to the pelvis, or by means of a considerable pedicle it is attached to the pelvic organs. If it is from the rectum or urinary bladder, it will usually give signs in the feces or urine before it becomes a prominent palpable tumor. When extragenital tumors have been excluded we may have considerable difficulty in deciding whether we are dealing with an inflammatory exudate, a blood extravasation, as from a tubal pregnancy, or an actual new-growth. The tumors of moderately large size are usually ovarian or parovarian in origin. Next in frequency are the subserous pedunculated fibromas of the body of the uterus. The diagnosis is usually easy until adhesions form, which may lead to an attachment of the tumor to some abnormally placed abdominal organ. It will then not be easy to exclude a pseudotumor or circumscribed tubercular exudate. Exploratory laparotomy will sometimes be necessary finally to establish the diagnosis. One of the important aids lies in the fact that the point of origin of the tumor, as well as the organ from which it springs, preserves its normal situation and its relations to the neighboring structures. Should a supernumerary or descending spleen be the seat of the tumor, the usual diagnosis will be of an abdominal tumor, although the origin will remain in doubt until established by operation. This is true also of tumors of the pancreas, usually. Still greater difficulty will be experienced when an ectopic, as a pelvic kidney, is the origin of the pathological process. The existence of multiple tumors is also a cause of failure in diagnosis. Since it is more likely that there is a single cause for the tumors in women, one thinks first of the ovaries as the seat of origin. Frequently both ovaries are involved. Echinococcus cysts, multiple ovarian cysts, and cysts of the mesentery cannot always be differentiated with certainty. Occasionally tumors of doubtful anatomical structure may present insuperable difficulties in diagnosis. These are usually retroperitoneal or between the layers of the mesentery. Sometimes these are myomas, sometimes fibromas, and they are frequently malignant. When the whole abdomen is filled by the tumor, and the abdominal organs are compressed together into a small space, an exact diagnosis will usually be impossible, and a laparotomy will be necessary to establish the actual circumstances of the case and to select the proper treatment.

The Extirpation of Malignant Tumors of the Tonsillar Region.—CLAIRMONT (*Zentralbl. f. Chir.*, 1907, xxxiv, 1393) says that resection of the lower jaw for the removal of tonsillar carcinoma which has extended to the surrounding region was recommended by Mikulicz and Kuster more than twenty years ago. Mikulicz divided the jaw 1.5 cm. anterior to and again 0.5 to 1 cm. above the angle, thus avoiding the attachments of the masseter and pterygoid muscles. Küster divided the bone in the line of the skin incision, which ran from the angles of the mouth

downward and backward across the masseter muscle to the anterior border of the sternomastoid. While this method permits an extensive exposure of the lateral pharyngeal wall, and almost to the end an extramural and pharyngeal operation, it results in a crippling and disturbance of the movements in the jaw. The portion of the jaw involved by the growth has been excised during the operation. In one case operated on by Kocher the jaw was divided in the median line, the muscles in the same line, and the mucous membrane alongside the tongue, with a resulting excellent exposure of the affected region. Recently good results have been obtained in cases in which a portion of jaw was resected, by the immediate introduction of a prosthetic apparatus to replace the resected portion. Clairmont operated on two cases by the Mikulicz-Kuster method, with the immediate replacement of the resected portion by a prosthetic apparatus. One patient died ten days after operation from an erosion hemorrhage from the common carotid. In the other case, five weeks after operaton, the temporary prosthetic apparatus was replaced by a permanent one. A preliminary tracheotomy does not seem to be necessary, because the patient is placed in a high position with an inclination of the head, so that the blood can escape easily from the wound during the operation, and its aspiration is thus avoided. The temporary prosthetic apparatus proves its value during the first days after the operation, in the fact that the patient can expectorate better than after the temporary splitting of the jaw with reunion of its fragments by sutures.

Experimental Generation of New Collateral Vessels for the Kidney.—GIRGOLAFF (*Zentralbl. f. Chir.*, 1907, xxxiv, 1444) calls attention to the excellent results obtained from Edebohl's decortication operation on the kidney, and to the claim that they are due to the abundant blood supply from the new-formed capsule. More recent writers have been inclined to consider that there are fewer vessels in the new-formed cicatricial capsule than were present in the normal capsule, and efforts have been made to replace the original method by a more efficient one. On the lower animals the decorticated kidney has been surrounded by omentum, and it is to this phase of the subject that Girgolaff devotes his attention. He performed a series of operations on cats, after the following method: Decortication and encapsulation in omentum. After one and a half, three, five, and eight weeks, the animals were killed and the bloodvessels were thoroughly washed out with salt solution through the thoracic aorta. The abdomen was then opened widely and the vessels of the kidney hilum and all adhesions of the kidney except the omental stump were divided between ligatures. The kidney was then attached merely by the omental attachment, and it was then injected with Berlin blue through the aorta. The colored material could reach the kidney only through the bloodvessels of the omental attachment. It was found to have reached the cortex and the medullary substance as far as the papillæ, and it could be seen macroscopically. The intensity of the injection was found to have increased with the interval between the time of the operation and the injection. He therefore concludes that a more abundant permanent collateral circulation was produced by grafting the omentum to the kidney than by Edebohl's method.

THERAPEUTICS.

UNDER THE CHARGE OF

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The Vasodilators in Cardiac Disease.—FRANZE (*Folia Therapeutica*, 1907, i, 73) states that the peripheral resistance in the arterial system is an important factor in the treatment of heart lesions. In the control of this resistance the several drugs most commonly used are glyceryl nitrate, amyl nitrite, sodium nitrite, and potassium and sodium iodide; the last two being most usually employed. They act by lessening the viscosity of the blood and thus diminish the resistance due to this factor. The preferable method of administration of the last is to give 1¾ to 5 grains five times daily for a period of two or three years; in every month a pause of one week, and after every three months a pause of a month, should be made. Other substances which may be employed to lessen high arterial tension are isobutyl nitrite, hydroxylamine hydrochloride, and diuretin, which last is to be preferred since it is practically free from undesirable by-effects.

Creosote Carbonate in Bronchial and Pulmonary Affections in Infants.—JOHN (*Therap. Monats.*, 1907, xxi, 516) draws the following conclusions from his observations of the action of this substance in 100 non-tuberculous patients: Creosote carbonate possesses a distinct curative action in the various bronchial and pulmonary affections; whether these be due to Fränkel's pneumococcus, the streptococcus, Friedländer's pneumonia bacillus, the staphylococcus, or to other causes. Whether this is due to its preventive influence upon bacterial growth or to the fact that it exerts a reparative action upon the local process or whether it combats the products of the growth of the bacteria in the organism it is not possible to do more than theorize. The results show that creosote carbonate is an agent of considerable efficacy, and one worthy of more extended use.

Calcium Chloride in Therapeusis.—CARLES (*Journal de médecine de Bordeaux*, 1907, xxxvii, 472) considers calcium chloride a most useful drug, and cites the following indications for its employment: It was first used in the control of hemorrhage, epistaxis, hemoptysis, etc., and in hemorrhoidal bleeding and hemophilia. Here it acts by increasing the coagulability of the blood. On account of its influence in the production of fibrin-ferment it possesses a favorable effect in urticaria and acute œdema; it is particularly useful in the urticaria resulting from the

injection of therapeutic sera and in that due to intoxication. Its antihemolytic action renders it useful in bilious hemoglobinuric fever. In nephritis it sometimes reduces the albuminuria, the reduction being due to its antihemolytic action, to its antitoxic effect, and to its antagonism to the sodium ion, the latter being increased in renal lesions; also in small doses it is diuretic. Its effect in lessening nervous excitability gives it a field of usefulness in tetany, glottic spasm, convulsions, and laryngismus stridulus. As a cardiac tonic it increases the force of the heart and lowers arterial tension, and in pneumonia it has a neutralizing effect upon the accumulation of the sodium salts in the organism. The drug is easily taken in doses of from 15 to 60 grains daily in milk, beer, or syrup of peppermint. It is important to interrupt its administration for one day in every four, and to suspend treatment every eight or ten days. In aged patients, who are liable to calcium retention, it should be avoided for its administration may result in calcification of the vascular system.

Mercury Bibromide in Syphilis.—DALIMIER (*Revue de thérap.*, 1907, lxxiv, 638) recommends the following formula as being particularly effective in the nervous lesions of syphilis: Mercury bromide, 27 grains; sodium bromide, 21 grains; sterile water to make 2½ drams. The mixture is employed as an intramuscular injection, and has the advantage that it causes no pain.

Vaccine Treatment in Skin Diseases.—WHITFIELD (*Journal of Cutaneous Diseases*, 1907, xxv, 529) considers that vaccine treatment foreshadows an enormous advance in our control of infective disorders, but our present lack of knowledge renders results uncertain in some instances. The vaccine treatment of boils is uniformly successful, and is the only reliable treatment for furunculosis. In sycosis the treatment is a valuable aid, but must be continued for long periods and is best combined with *x*-ray depilation. In acne the treatment is uncertain, sometimes proving successful and at others of no avail. In septic dermatitis and ulcers the treatment is valuable as an auxiliary. In Bazin's disease it is uncertain, but sometimes of assistance. It is of great value in tuberculous ulceration. In lupus the treatment alone is too slow and uncertain to be recommended, but may prove of value in preventing relapse after treatment with Finsen's light, and it may be combined with benefit with the *x*-rays.

The Treatment of Laryngeal Tuberculosis.—JURASZ (*Deut. med. Woch.*, 1907, xxxiii, 1073) recommends in the prophylaxis of this condition the inhalation of sprays of alkaline or sodium chloride solutions, the employment of expectorants and balsam of Peru, and disinfection of the sputum. In local treatment the astringents should be avoided, but insufflations of boric acid, orthoform, or iodol may be employed. The infiltrations of the posterior wall of the larynx and of the interarytenoid fold may be curetted and cauterized with 30 to 80 per cent. lactic acid; the galvanocautery may also be used here. The infiltrations of the arytenoid cartilages should be scarified. The direct action of the sun's rays and treatment by Bier's hyperemia, especially the latter, give

good results in instances of pain. Tracheotomy, performed to relieve dyspnœa, also benefits the local condition. Perichondritic inflammations may necessitate incision, and in involvement of the epiglottis ablation may be indicated.

The Treatment of Enteric Fever.—TUTSCH (*Allg. Wien. med., Zeitung*, 1907, lii, 333, 343) has treated enteric fever for the past five years in the following manner: While the certainty of the diagnosis is not assured the patient receives 7½ grains of quinine hydrochloride daily. When the diagnosis is certain this is replaced by an emulsion of 3 drops of creosote carbonate, 3 drops of eucalyptol, and about ½ ounce of boiled milk or wine of condurango; this is given thrice daily. Twenty-six patients thus treated recovered without complications, and their symptoms became rapidly ameliorated. The course of the disease usually lasted about four weeks, but in a few instances the temperature fell before this time had elapsed.

A New Treatment for Diabetes Mellitus.—SOULES (*New York Med. Jour.*, 1907, lxxxvi, 929) reports a patient affected with diabetes mellitus in whom he has achieved a cure by means of the administration of the fluidextract of chimaphilia, 2 drams in a glass of milk at each meal-time. The drug acted best in connection with an antidiabetic diet. As soon as the sugar disappears from the urine it is advised that arsenic be added to the medication in doses gradually increased to medium dosage. This should be continued for at least two months, and if the sugar does not re-appear starchy diet may be very gradually resumed reducing the chimaphilia one-half. If the glycosuria re-appears the starches should be omitted and the full doses resumed. After a few weeks the starches may be again allowed with caution and so on until the patient is cured.

Santonin in Diabetes.—According to BERNARD (*La quinzaine thérap.*, 1907, viii, 415) the treatment of diabetes by santonin gives constant results in all types of diabetes. In the mild instances about $\frac{3}{10}$ grain, combined with a vegetable cholagogue, is given twice daily with the principal meals; in the more severe forms a third dose is added at breakfast time. This medication is continued for three weeks and is followed by an intermission of from one to two weeks during which the patient's hygiene is regulated and his diet restricted. Other courses of santonin treatment are instituted depending on the persistence of the glycosuria. The alkalies are entirely compatible with the santonin and afford a useful adjunct to this form of treatment.

The Prolonged Administration of Digitalis.—ACHERT (*Berl. klin. Woch.*, 1907, xliv, 1115) considers digalen (soluble digitoxin) the preparation to be preferred in order to avoid cumulative effects. It is especially useful in myocarditis, cardiac weakness of chronic type, and in the cardiac disorders which occur in chronic nephritis. The ordinary dosage is from 7 to 14 drops of the solution in which the drug is marketed; these quantities may be administered for continued periods in the above affections without causing unpleasant by-effects.

The Opsonic Treatment of Tuberculosis by Tuberculin.—TURTON (*The Practitioner*, 1907, lxxix, 650) reports results of the treatment of seventeen patients by this means, and summarizes as follows: Seven of the patients did very well and four others showed most encouraging improvement as a result, in his opinion, of the tuberculin injections. Other patients were less favorably affected. Of the causes of failure probably the first was that the treatment was not begun early enough in the diease; here the immunizing power may be exhausted, the general nutrition at a low ebb, and the tissue destruction so great that recovery is hardly possible. Economic conditions also often interfere with success, for instance some patients cannot be removed from unsanitary surroundings, and it may be impossible to procure proper food for them. Failure also may be due to the fact that it is impossible to inject, in each instance, a specific tuberculin manufactured from the particular strain of tubercle bacilli. In other than tuberculous affections the greatest advantages are to be obtained by injecting the patient with a vaccine made from his own infective organisms, and possibly greater success might be attained from a similar process in tuberculosis. Another bar to success in this form of treatment is the presence of a mixed infection. The doses used varied from $\frac{1}{400}$ to $\frac{1}{2000}$ milligram of a tuberculin solution diluted with 0.5 per cent. phenol so that 1 c.c. contains $\frac{1}{500}$ mg. of solid substance. The periodicity of the injections varied from four to twenty-one days. He is not inclined to abandon the use of the opsonic index in carrying out the treatment, but considers careful attention to the patient's reaction, as shown by the index, to be essential to successful vaccine treatment.

Tuberculotherapy in Tuberculosis.—ROSS (*New York Med. Jour.*, 1907, lxxxvi, 869) advocates the feeding of tuberculous patients with the raw or partially cooked meat from tuberculous animals. Such meat contains the toxins and antitoxins of the animals in proportion as their blood serum has had the power, and the muscular tissue and blood serum of a tuberculous animal are the main sources of that animal's resistance; and so their flesh possesses immunizing qualities, hitherto not deliberately utilized to cure disease. The exhibition of raw meat to phthisis patients is an inexact process since meat from a tubercle-free animal and meat from a tuberculous animal are mixed from day to day. It is only by obtaining tuberculous meat and administering it in an exact and scientific manner that the full benefits in which Ross believes can be obtained. Accurate methods which he describes enable one to administer to a patient a measured quantity of meat containing tuberculotoxins and antitoxins, and to observe their reactions with some degree of accuracy.

Antituberculosis Serum and the Tubercle Bacillus.—BINSWANGER (*Zentralbl. f. die gesammte Therapie*, 1907, xxv, 454), in reviewing Marmorek's work and that of others on this subject, states that tuberculin has given very satisfactory results in the treatment of bone and joint tuberculosis, but that in the pulmonary form of the infection its action has been much less effectual. This is explained on the ground that in

pulmonary tuberculosis there is usually present a mixed infection which militates against the beneficial effects of the tuberculin. On this account Marmorek immunized his "antituberculosis horses" against the streptococcus, thus producing a "double serum." The statistics of this serum are, however, at present too few to allow of definite conclusions being drawn.

Specific Action of Moebius' Antithyreoidin.—HOLST (*Klinisch-therap. Woch.*, 1907, xiv, 1084) is firmly convinced that this preparation possesses a specific action in exophthalmic goitre. To strengthen his assertion he reports a patient who was suffering from the disease in its typical form and in whom the iodine treatment had no effect save to induce rhinorrhœa and acne, but who immediately began to improve under treatment by antithyreoidin. The goitre became smaller, the palpitation, tremors, exophthalmos, and headache less, and in about six weeks the treatment was stopped and the patient was discharged. Two months later she reported that there had been no recurrence. Holst considers the fact that the severe headache from which the patient suffered, and which had been rebellious to all forms of antineuralgic and counterirritant treatment, disappeared under the administration of the antithyreoidin is a strong point in favor of the specificity of this agent. The dosage which the patient received varied from 5 to 30 drops daily.

PEDIATRICS.

UNDER THE CHARGE OF

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Bacteriological Infection of the Urinary Tract in Childhood.—C. R. Box (*Lancet*, 1908, i, 77) classifies infections of the urinary tract in children as descending and ascending infections, and those by contiguity. Descending infection is also termed hematogenous, and is not by any means a common occurrence. The body can dispose of enormous numbers of bacilli (proved by injection) without their appearing in the urine or other secretions. When they do appear, the time of their appearance and the substances which appear with them lead to the presumption of renal damage. The quantities which appear in the urine are not in any way comparable to the quantities injected. Bacilluria is usually due to an ascending infection, and the ureter is the common highway of infection. The commonest infection in children is due to the colon bacillus. The more frequent occurrence of these infections in girls than in boys is due to the shortness of the female urethra and the proximity of its orifice to the anus. The colon bacillus is frequently found in the urine of children suffering from thread worms; the ova and the worms even have been found there, thus suggesting that they may carry the colon bacillus into the urine and even the bladder.

The clinical manifestations of infection of the urinary passages in childhood may be pyelitis, cystitis, or incontinence of urine without obtrusive evidence of local inflammation. Pyelitis may occur as a febrile affection with sudden onset, chills, and pyrexia; the possibility of its presence should always be borne in mind when dealing with cases of obscure fever in childhood, whether of a sustained, remittent, or relapsing character. Shivering and rigors especially should direct attention to the urinary tract. Gastro-intestinal disturbances, pneumonia, tuberculosis, and malaria may be closely simulated. Cystitis of a more or less acute character is not uncommon in childhood; no definite cause is usually apparent; the fever is usually very high, although mild attacks may also occur. Incontinence of urine may be associated with bacilluria; the organism is almost invariably the colon bacillus. A trace of albumin is often present in these cases, but casts are always absent. The cases are often associated with slight elevation of temperature at night. The cystitis of childhood when due to the colon bacillus demands a guarded prognosis. Acute cases improve usually, others less acute drag on indefinitely. The preventive treatment includes frequent bathing and the avoidance of contamination from the anus. Vulvitis must be promptly treated. Mild cases of pyelitis, cystitis, etc., require full doses of citrate of potassium with belladonna. In severe cases thread worms must be carefully looked for and treated if found; drugs have been invariably disappointing; serumtherapy led to improvement in only one case.

Precocious Menstruation.—W. Stoelzner (*Med. Klinik*, 1908, iv, 5) reports the case of a girl, aged two years and ten months, who had menstruated since her twenty-fourth month. At first the intervals between menstruations were eight weeks, but now they are only five weeks. The breast development is that of a girl fourteen years old, and the vulva, uterus, and other internal genitalia (examined per rectum, although the vulvar orifice was large enough to admit the finger) also showed a remarkable development. There was no hair development. At birth, and for some time after, the child appeared perfectly normal, but at the present time the general features of the child give it the appearance of being at least five years old.

Estivo-autumnal Fever in a Child Two and a Half Years Old.—R. A Clock (*Archives of Pediatrics*, 1907, xxiv, 918) reports the case of a girl aged two and a half years, who had been perfectly well until suddenly she complained of abdominal pain and vomited; the vomiting was repeated five times. Marked symptoms of pulmonary congestion were present, which, however, soon disappeared. Remittent fever (varying 3° to 4° daily) was present, but only a moderate increase in respiration and pulse rate. On the fourth day the spleen was found enlarged and easily palpable. The temperature range on this day was 6°; there was no chill, no sweating, no cyanosis, no pallor, no discomfort outside of marked restlessness and peevishness. The finding of malarial plasmodia on the seventh day established the diagnosis; there were hyaline bodies, crescents, signet-ring shaped organisms, and one ovoid. Aristochin in 40 grain doses daily checked the process at once. The

following points are emphasized by the author: The marked pulmonary symptoms present at the onset of the disease, masking the real infection; the absence of typical malarial symptoms; the prompt and permanent action of aristochin; the relatively infrequent occurrence of the disease in this latitude in so young a child; and absence of any positive infection.

Malnutrition in Older Children.—A. FRIEDLAENDER (*Lancet-Clinic*, 1908, xcix, 53) writes of malnutrition in older children independent of primary organic disease. Such children are more liable to infections, and should, therefore, especially as there are very many, be given considerable attention. Apart from their physical deficiency such children are frequently easily picked out because of their mental precocity. Of the etiological factors in malnutrition heredity is probably the most important. The parents of such children have delicate constitutions, they may have tuberculosis, gout, syphilis, or be alcoholics. The children inherit a highly developed nervous system with a corresponding amount of physical deterioration. Other causes are improper feeding in infancy, bad hygienic surroundings, especially a lack of fresh air due to fear of "catching cold," education at the expense of physical well-being, too much society and insufficient amount of sleep, too many after-school lessons (music, drawing, dancing, etc.). The characteristics of such children are carefully described by Friedlaender. He emphasizes their tendency to catarrhal inflammations of the mucous membranes; their vulnerability is very striking, and slight causes in them produce very marked effects; gastro-enteritis may be brought on by a slight deviation in diet, a long, persistent coryza from slight exposure to dampness. The treatment should include regulation of diet and general hygiene; regular meal hours, three meals per day; nothing between meals, especially no candy or other sweets. The food should consist of meat, eggs, milk, vegetables, cooked cereals, bread, butter, fruit, and light cakes. A daily afternoon hour for rest and an early hour for bed should be insisted on. Outdoor exercise is necessary, and several hours daily are not too many. The windows should be kept open, more particularly at night. Cold sponging is only suitable in children who react readily from the shock. Mental overexertion is to be curbed. Medicinal tonics are suitable; the iodides are excellent alteratives, especially in recurrent bronchial attacks.

The Bacteriology of Meningitis.—F. S. CHURCHILL (*Archives of Pediatrics*, 1907, xxiv, 881) has studied 26 cases of meningitis in children from the standpoint of its etiology, the extent of variation of its organisms, the manner in which the organisms gain access to the system, the difference between the different types of organisms and the prognosis in the various forms of the disease. His cases include 3 of tuberculous meningitis. He found that meningitis in early life is caused by a great variety of microörganisms, chief among which in frequency are Diplococcus intracellularis meningitidis, or the meningococcus of Weichselbaum, and the pneumococcus of Fraenkel. A large proportion of cases, especially in epidemics, is due to the menigococcus. A considerable number of cases, however, was due to the pneumococcus, variously estimated at from 11 to 22 per cent. in epidemics, and higher in sporadic cases. Mixed infection he found very rarely. He found the meningo-

coccus very wide-spread in its distribution in the body. It gains access to the system through the nose and throat. It produces a true meningococcic septicemia. He found the meningococcus in the nose and throat of healthy individuals living in close contact with meningitic cases of this type. Such individuals should be isolated, as by their presence abroad they help to spread the disease. The type of meningitis cannot be determined from the clinical picture. This can be done only by lumbar puncture. The prognosis in the pneumococcic variety is more unfavorable than in other types. Meningitis due to almost all the known pathogenic bacteria does occur, but is infrequent. As the meningococcic is the only type which is really contagious, the throat and nose and also the spinal fluid of all cases should be examined to determine the variety present in order to protect the community. The meningococcic cases certainly should be quarantined, possibly also the pneumococcic.

OBSTETRICS.

UNDER THE CHARGE OF

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Operative Treatment of Intracranial Effusions of Blood in the Newborn.—SEITZ (*Zentralbl. f. Gyn.*, Nr. 30, 1907) reports the case of a primipara with normal pelvis, who gave birth to twin children. The first was born spontaneously; the second was expelled spontaneously thirty hours after the first, in the second position, vertex presentation, the expulsive period of labor lasting five hours. The child was not asphyxiated at birth. Over the left eye and side of the face there was œdema. There was a caput on the occipital bone; the parietal eminences seemed to be forced outward, and the greater fontanelle was wider than normal and also the frontal suture. The bony development of the cranium seemed normal.

At first the child was quiet and took nourishment, slept, had no vomiting, and the mother noticed nothing unusual about it. Early on the third day the child was quiet, but was noticed to be pale, with very slight jaundice. The greater fontanelle was very tense and distended and on palpation rhythmical beating could be heard; this was synchronous with the heart. The breathing was regular eight to ten times in each fifteen seconds, occasionally becoming slightly irregular, and was reasonably deep. The skin of the child was slightly mottled, and the sagittal suture somewhat wider than normal. The right leg of the lambdoid suture was wider than the left, and over the right parietal eminence the veins were distended as far as the right ear, while a vein on the left side between the forehead and the vertex was also distended. The child seemed perfectly apathetic, turning the head more toward the left, but lying as it was placed.

The child would open the left eye widely from time to time, while the right remained closed. The eyes were turned toward the right; the left could be easily opened, while the right was held tightly shut. The left corner of the mouth was a little higher than the right, but when the child cried the retraction of the left side was not seen. Evidently the upper portion of the facial nerve was paralyzed. The left pupil was very tightly contracted and the right considerably wider. When the child's head and face were tapped suddenly over the facial nerve a convulsive movement followed; the head was drawn to the right, the left sternocleidomastoid muscle contracting; the right was drawn in a tonic spasm; the left eye was one-half open, the right held tightly shut, and both eyes were directed toward the right and below; the forehead was wrinkled; the breathing became irregular when the child cried. After such an attack the left eye remained more widely open. The child's neck moved freely; the back was somewhat stiff, the left arm easily contracted. There was no difference in the reaction of the legs. The left patellar reflex was more than the right. During the day the condition of the child became worse. The left eye remained widely open; the inferior portion of the facial nerve showed no especial paralysis; the right pupil dilated and the breathing became shallow and irregular. There were no convulsive efforts at respiration, but the child was pale, slightly jaundiced, the temperature subnormal; while if the child's body was pressed upon œdema easily developed. The greater fontanelle was widely distended, and lumbar puncture gave 1 c.c. of slightly blood-stained fluid. The diagnosis was made of intracranial hemorrhage with pressure, and it was thought that the bleeding was situated in the left optic thalamus. The great alteration in the breathing led to the belief that an accumulation of blood had formed beneath the tentorium. The child's head was prepared by shaving and an incision made on the right side extending in a curved form over the region where the hemorrhage was suspected. The vessels in the incision did not bleed with especial freedom. The periosteum was separated over the bone through the extent of the incision 1 cm. in width. Beginning at the external lateral border of the greater fontanelle, the edge of the bone was freed with the knife over the extent of 3 cm. The dura was separated from the bone and opened with blunt scissors in such a manner as to turn the entire right parietal directly outward. The dura was not especially tense and showed a pale-blue color. The dura was incised with the point of the knife, when blood, in a considerable stream, spurted out. The incision was then enlarged, the clot removed, and an effort made to remove entirely the blood by sponging with gauze. The hemisphere was then irrigated with salt solution, the dura closed with catgut stitches, the parietal bone replaced, and the skin incision closed. An antiseptic dressing was applied and by adhesive plaster made firm over the whole area. The operation was done without anesthesia and the child was comatose in the beginning. After the dura was incised the child breathed more freely and cried. The left eye was closed, but there was no evidence that the relief of cerebral pressure influenced the sensibility of the child. As the infant was anemic through loss of blood, 50 c.c. of salt solution were injected beneath the skin. The child was then placed in an incubator. The operation had consumed

twenty-five minutes. During the next few hours the left eye again became closed, the child passed into coma, took no nourishment, had subnormal temperature, and died ten hours after the operation. Autopsy showed that the left hemisphere was entirely free from blood, but over the right, in the posterior and anterior portions, there were small coagula, but that the large clot had been removed by operation and there had been no bleeding afterward. In the region of the cerebellum the tentorium was found distended with blood which reached to the medulla. This blood could not be removed through incision and was the cause of the death.

Seitz further reports the case of a primipara with a normal pelvis, delivered in spontaneous labor, the presentation and position of the child being normal. Heart sounds during the latter part of labor were good, the pains were not especially strong, and the head passed rapidly through the birth canal. The body was not delivered at once and the child became cyanotic and required assistance in the delivery of the shoulders. Respiration and crying occurred naturally and there was no discharge of meconium in the amniotic liquid. The child was a male of normal proportions. The bones of the head were somewhat hard, they were forced over each other to some extent, and the greater fontanelle was wider than normal; the caput small. Immediately after labor the child showed nothing especial. It nursed naturally and cried during the night and continued during the following few days to be apparently well, except that the mother noticed some jerking movements of the head. On the morning of the second day, forty-two hours after labor, the child had a sudden and severe attack of cyanosis, for which it was immediately placed in a warm bath. These attacks were repeated, the right leg and arm becoming spasmodically contracted; the arm was slightly flexed, the hand drawn inward and also the thumb and finger. The left side showed decided stiffness, but there were no spasmodic contractions. The greater fontanelle was not tense. The head was directed most of the time toward the right; the lids of the left eye were closed; the right eye could easily be opened. Both were directed downward, showing only the whites. There were convulsive movements about the mouth and eyes. The child seemed much improved by a prolonged warm bath, and efforts were made to assist the respiration by passive movements. Lumbar puncture gave 1 cm. of a faintly bloody fluid. The child became worse and died, the body before death becoming covered with bluish-red blotches, the skin of a grayish-bronze appearance. Death ensued forty-eight hours after birth. Upon autopsy the dura was loosened from the bones over the right and left parietal eminences. When the dura was opened it was found that the vessels of the pia mater were greatly distended with blood, the veins appearing like thick cords, with very slight extravasation of blood from the vessels. The longitudinal sinus was greatly distended. The tentorium was distended with blood, so that both hemispheres of the cerebellum and medulla were covered with fluid blood. The ventricles were free from blood; and death in this case occurred from the accumulation of blood beneath the tentorium.

Roentgen-ray Examination after Pubiotomy.—FRANK (*Zentralbl. f. Gyn.*, 1907, Nr. 30), after two cases of pubiotomy, studied the condi-

tion of the pelvis by the Röntgen rays. His first case was that of a woman with a generally contracted pelvis, with a true conjugate of 8 cm. The pelvis was opened with a saw and the child safely delivered with forceps. The second patient had a flat pelvis, with true conjugate of 9 cm. There was a laceration of the vagina connecting with the wound in this case. The wounds were sutured and drained with iodoform gauze, the patient making a good recovery. In both these cases the Röntgen rays revealed a fibrous union between the bones. There seemed to be no effort to form bony tissue.

Abortion Produced by the Roentgen Rays.—FRAENKEL (*Zentralbl. f. Gyn.*, Nr. 31, 1907) reports the case of a tuberculous patient, in whom it was determined to interrupt pregnancy with the hope of checking the tuberculous process. Twenty-five applications of the Röntgen rays were made, each lasting from five to ten minutes. After each five days a pause of one day was allowed to intervene before new applications were made. The region over the ovaries and the thyroid gland were chosen for the applications. Especial care was used to avoid injury to the skin. Spontaneous abortion occurred with free hemorrhage, which ceased when the uterine contents were expelled. No vaginal examination was made during the case, so that the effect is attributed entirely to the Röntgen rays.

GYNECOLOGY.

UNDER THE CHARGE OF

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Primary Fibro-myomas of the Broad Ligaments.—JAMES VANCE (*Annals of Surg.*, 1907, xlvi, 854) reports a case in which the presumptive diagnosis was the same as this title. He comments on the divergence of opinion as to origin of the fibromyomas found in the broad ligament apparently unattached directly with the uterus, admitting the preponderance of evidence and opinion is that they spring from the uterus, and then proceeds to substantiate his belief to the contrary. He admits such broad ligament origin is very rare, stating that in American medical literature of the last eleven years but four cases of true broad ligament fibromyomas with normal uterus have been recorded, but presumes to demonstrate these were not of uterine origin.

It is very probable that all fibromas and fibromyomas lying just without and immediately connected with the pelvic peritoneum are of uterine origin. Exceptions to be noted are those springing from the ureter, bladder, ovary, Fallopian tube, and rectum and retaining direct connection with the site of origin. The subject of migrant and parasitic tumors of the pelvis has been carefully studied by Cullen, Stone, Douglas, and others, not alone from the view-point of literature, but from the

clinical and pathological aspects. Their structure in no way varies from that of the same variety of neoplasm originating in the uterus, while primary growths of the broad ligament might be expected to contain striated muscle fiber, inasmuch as such tissue exists in the broad ligament and not in the uterus. Moreover, when we must conclude that mucoid neoplastic inclusion of portions of the endometrium of Gaertner's ducts is the sole cause of mucoid degeneration of uterine growths of this nature and the existence of islands of mucosa within them, together with epitheliomatous transformations thereof, we have reason to believe neoplasms of broad ligament origin should at least occasionally contain structures that do not exist in the uterine body, but none such have ever been recorded.

The Treatment of Pelvic Infections.—C. W. BARRETT (*Annals of Gyn. and Ped.*, 1907, xx, 488) discusses this subject in a lengthy paper. He emphasizes the fact that the disease is an infection and that the inflammatory process is not a disease but an attempt of nature successfully to resist the onslaught of the infection. Barrett declares that pelvic inflammation has not the power of self-extension, but rather tends to repair; that during the acute stage of a pelvic infection operative intervention should not be employed until determination that drainage or removal of some organ will remove or decidedly lessen the source of infection, when drainage, or the removal of the uterus or a tube or an ovary, may be clearly required; for the inflammation, *per se*, the removal of acutely inflamed pelvic organs is not indicated; non-puerperal, non-operative, acute infection is usually, but not universally, of gonorrhœal origin; and that an acute primary infection of the appendages should receive early conservative treatment, although drainage to save life or hasten convalescence is often required. Barrett advises removal of chronic pus tubes, but in the presence of active infectious processes advises vaginal incision and drainage as a preliminary procedure, a radical operation to follow when necessary after the disappearance of the acute infection.

The Renal Catheter as an Aid in the Diagnosis of Valve-like Obstruction of the Ureter.—J. A. SAMPSON (*Annals of Surg.*, 1907, xlvi, 870) describes two interesting cases in which use of this instrument was relied upon for diagnosis, and in one of them the diagnosis was confirmed by operation. By coating the catheter with wax Sampson was able to locate multiple constrictions. To the inexperienced, and but few can lay claim to experience in ureteral catheterization, passing of ureteral catheters may seem to be a quite simple procedure. Perhaps those having a fairly large experience in surgery of the kidney and ureter, with some but less experience with the various mechanisms employed as aids in diagnosis, there appears a better estimate of the practical value of such so-called instruments of precision. For example, there is probably not on record a case of fused kidney diagnosed before operation by these methods, although it is evident catheterization of the uteters in such condition has been attempted.

Instrumental Perforation of the Uterus.—KUNTZSCH (*Zentralbl. f. Gynäk.*, 1907, xxxi, 1590) comments on a number of cases of perfora-

tion of the uterus which have come under his observation. Some of these were due to the use of a curette by skilful operators, which leads him to think that many accidents of this kind are to be attributed to the instrument used or to the conditions of the uterus rather than always to want of skill, as frequently stated. Kuntzsch calls attention to three cases in which laparotomies were performed and scars found in the fundus of the uterus, indicating old perforations. The history of these cases showed that a curettage had been done but that no symptoms suggestive of perforation had been observed. He thinks, therefore, that perforations of the uterus are more common than is generally believed.

Operative Treatment of Purulent Parametritis.—C. FRANCKE (*Zentralbl. f. Gynäk.*, 1907, xxxi, 1597) describes a large abscess of puerperal origin that was drained through a small incision made above Poupart's ligament. The opening failed to close for a year, when an operation was performed, the fistulous tract being removed and a considerable portion of the ileum resected. The abscess cavity was thus obliterated and the result of the operation was successful. In similar cases in which the abcess walls can not contract to obliterate the cavity Francke thinks that there should be no hesitation in making an extensive resection of the ileum.

Ventral Fixation of the Round Ligaments.—P. RISSMANN (*Zentralbl. f. Gynäk.*, 1907, xxxi, 1588) sutures the round ligaments to the abdominal wall, leaving the uterus entirely fee. Three silk sutures and sometimes one or two catgut sutures in addition are passed through each round ligament at a distance of 4 to 5 cm. from the uterus. Rissmann has followed this procedure for five or six years and has not seen a relapse.

An Unusual Parovarian Tumor.—A. CZYZEWICZ, JR. (*Zentralbl. f. Gynäk.*, 1907, xxxi, 1583), states that nearly all tumors of the parovarium are unilocular, intraligamentous cysts. In a case reported by him two dermoid cysts about the size of goose eggs were removed from a woman aged forty-one years. Both tumors were inserted in the posterior layer of the broad ligament. One had its origin clearly in the ovary. The other, which was pedunculated and located between the ovary and and uterus, originated evidently from the parovarium or a supernumerary ovary. As no trace of ovarian tissue was found in the cyst or its pedicle, Czyzewicz concludes that the second tumor was a dermoid of the parovarium.

End Results in Operations for Prolapsus Uteri.—J. W. KEEFE (*Amer. Jour. Obst.*, 1907, lvi, 616) refers to the lack of unanimity of opinion as to the best method of treatment of prolapsus uteri, and then gives the results he has obtained by means of the Alexander operation of shortening the round ligaments combined with plastic surgery upon the cervix and vagina. Keefe says intra-abdominal pressure and increased weight of the uterus are important factors in the production of this condition. His experience is in 48 cases, and of these, 46 were treated by the combined methods of plastic operation on the parturient canal and the Alexander operation. Two were subjected to plastic operations first,

and upon one of them the Alexander operation was done. In one of these the return of prolapse followed parturition four years subsequent to the plastic operation. The other of these two bore two children after the Alexander operation, which left her with moderate cystocele and rectocele but no prolapse. Keefe has traced 23 of the 48 cases, and in 18 of those traced the uterus is in good position and the previous symptoms are gone. In 2 of the remaining 5 cases there was a return of the prolapse to the second degree, but without any subjective symptoms. The other patient in which failure was recorded was advised to have hysterectomy done. Seven have borne children since operation, 2 of them having borne two each. Keefe's best results were in complete prolapse. Of 19 cases of this degree, he subsequently examined 12 without being able to find a complete failure in any case in which the Alexander operation was done. One failed when subjected to plastic operation alone; but after an Alexander operation was done the success was complete. Two cases had a return of the prolapse to the second degree without subjective symptoms.

Phlebitis Following Abdominal Operations.—O. G. PFAFF (*Amer. Jour. Obst.*, 1907, lvi, 630) refers to the increased coagulability of the blood late in typhoid fever and of various other interesting matters, from their etiological relation to postoperative phlebitis. Pfaff thinks many of the cases of phlebitis are simply extensive aseptic blood clots, without any true inflammation, and that one important condition in the causation of postoperative phlebitis is an abnormal plasticity of the blood. Pfaff also thinks the clot generally receives a mild form of infection introduced into the wound during the operation which later extends to the walls of the veins. Believing stagnation is a very important element in the etiology, he suggests the adoption of getting patients up early after operation, as has for years been strongly urged by Emil Ries and more recently by Boldt. Pfaff further states that the very high state of plasticity of the blood being an etiological factor, coagulability should be in every instance determined before operation and, if abnormal, be brought down to normal before the operation is done.

DERMATOLOGY.

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The Etiology of Impetigo Contagiosa.—BENDER (*Archiv f. Dermat. und Syphilis*, 1907, Band lxxxiv, Heft 1–3) after a careful bacteriological study of a number of cases of impetigo contagiosa, concludes that the

following may be regarded as established, although the actual cause of the disease is not yet determined. (1) The ordinary staphylococci are certainly not the cause of impetigo contagiosa. (2) Streptococci are with the greatest probability to be regarded as the cause of this affection. (3) The impetigo cocci first described by Unna, are not common staphylococci; they are no nearer related to these than to streptococci.

The Etiology of Erythema Nodosum.—HILDEBRANDT (*Münch. med. Woch.*, Nr. 7, 1907) reports the case of a woman, showing signs of tuberculosis as a child, who was seized with an acute angina, stomatitis, and, after ten days, an erythema nodosum. At the acme of the attack there was a positive diazo reaction, and two guinea-pigs inoculated with venous blood of the patient intraperitoneally died of tuberculosis. The author accordingly concludes that, at the height of the erythema nodosum, virulent tubercle bacilli were present in the circulation. This patient subsequently suffered, first from a right-sided, then from a left-sided pleurisy, and later from pericarditis. The author refers to 4 other cases of erythema nodosum in which there were the signs of beginning pulmonary tuberculosis, and expresses his belief in the possibility of a form of disease being produced by the tubercle bacillus which may be distinguishable from ordinary erythema nodosum.

Carbolic Acid in Diseases of the Skin.—KROSING (*Archiv f. Dermat. und Syphilis*, Band lxxxiv, Heft 1–3) finds carbolic acid an excellent and harmless remedy in suitable diseases of the skin, being especially indicated in furunculosis and other affections due to pyogenic micro-organisms. It is most useful in alcoholic solutions varying in strength from 20 to 50 per cent. Treatment with carbolic acid is not as well suited to large furuncles as to the small ones which occur upon the nape of the neck and in the anal and scrotal region, where frequently a single thorough application of a strong alcoholic solution is sufficient to cause the disappearance of the furuncles. In many cases of sycosis vulgaris repeated pencillings with a 20 per cent. to 50 per cent. solution will produce a rapid cure. In a chronic case of suppurative folliculitis of the vibrissæ the author has seen an immediate and lasting cure follow this treatment. Kerion celsi is another affection favorably influenced by this remedy. Impetigo contagiosa is quickly cured by applications of carbolic acid, but reddening and pigmentation are apt to remain for a longer time after such treatment than after the employment of other remedies; for the same reason the author has given up its employment in acne.

A Special Form of Trichophytosis as a Sequel of the Permanent Bath.—JACOBI (*Archiv f. Dermat. und Syphilis*, Band lxxxiv, Heft 1–3) describes an unusual form of dermatomycosis observed in patients treated by the permanent bath, the majority of the cases occurring in the psychiatric clinic at Freiburg. The affection began usually, but not always, in the region of the genitalia, frequenty also in the axillæ, as dark red, slightly infiltrated papules, which scattered about in great numbers, spread over a great part of the body, involving finally all parts

coyered permanently by the water. The face also showed single plaques, but only on the side which was mostly under water. In highly developed cases the palms and soles were affected, but it was never seen to attack the hair nor nails. There was neither itching nor pain. In large patches three zones could be distinguished. A very slightly elevated, bright-red border; a dark-red, livid centre, and between the two a narrow ring of thickened, whitish, opaque epidermis. In the whitish accumulations of epidermis an enormous quantity of mycelial threads could be demonstrated which were double-contoured, shining, interlaced, and branched, homogeneous, and without septa. The author concludes that this form of trichophytosis is produced by a germ normally present in the bath water which finds in the greatly macerated epidermis of cachectic individuals conditions favorable to producing a mild degree of virulence, a virulence which is completely lost upon drying.

A Case of Paget's Disease Treated by the X-rays.—G. W. DAWSON (*Brit. Jour. of Dermatology*, June, 1907), at the April meeting of the Dermatological Society of Great Britian and Ireland, presented a case of Paget's disease of the breast in a woman, seventy-nine years old, who for six months had been submitted to *x*-ray treatment. The treatment had produced a very manifest improvement.

Healing of a Carcinoma by Sunlight.—WIDMER (*Münch. med. Woch.*, 1907, Nr. 13) reports a case of carcinoma of the back of the right hand accompanied by painful swelling of the neighboring glands, occurring in a woman eighty-one years old, in which healing was obtained by exposure for a prolonged period to the rays of the sun. Exposures four hours long were made almost daily for three months. The author has likewise seen healing follow this method of treatment in an unusually short time in ulcers due to frostbite, chronic leg ulcers, chronic fistulæ following suppuration of the glands of the neck, eczema, bed-sore, and herpes tonsurans. The reflected rays are regarded as especially therapeutically active.

PATHOLOGY AND BACTERIOLOGY.

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Observations upon Paroxysmal Hemoglobinuria.—In previous papers DONATH and LANDSTEINER have shown that the blood serum of patients suffering with paroxysmal hemoglobinuria contains, during the attack, a substance which unites with the red blood corpuscles at low temperatures and on subsequent heating at 37° C. in contact with normal serum

causes their hemolysis. These observations have been confirmed by others. Widal and Rostaine have offered an explanation for the phenomenon which differs from that of Donath and Landsteiner. They consider that the normal serum contains an antihemolysin which, in cases of paroxysmal hemoglobinuria, is diminished in amount and therefore allows auto- or iso-hemolysis to take place. DONATH and LANDSTEINER (*Cent. f. Bakt. und Parasitknd.*, 1907, xlv, 205) now find that the antihemolytic action of the normal heated serum is no greater than that of the serum from cases of paroxysmal hemoglobinuria.

It was further shown that in some rabbits the blood serum had the same property of making the red blood corpuscles, when brought in contact with them at low temperatures, susceptible to hemolysis by small amounts of normal guinea-pig's serum at 37° C.

They therefore conclude that their original supposition was correct. Since 3 of 5 of their cases of paroxysmal hemoglobinuria gave a history of syphilis, the authors suggest that this toxic autolytic substance may in some way be produced by a syphilitic infection.

CHOROSCHILOW (*Zeit. f. Klin. Med.*, 1907, lxiv, 431) too, in a short series of experiments in one case of paroxysmal hemoglobinuria, has not been able to confirm the opinion which Widal and Rostaine expressed. In two cases which Choroschilow studied, he performed the following experiments: The fingers of one hand of the patient were kept for several minutes in ice cold water. Then blood was drawn from the chilled fingers, and for control from the fingers of the hand which had remained at room temperature. When the blood from the chilled fingers clotted the serum which separated out was seen to be quite deeply tinged with hemoglobin. The serum above the clotted blood taken from the fingers of the other hand, although showing some hemoglobin, was never so deeply colored. Control experiments carried out in the same manner with normal individuals never gave the same results. The serum was not discolored. He found, however, when the blood from the cases of hemoglobinuria was prevented from clotting, that visible hemolysis did not occur and the supernatent plasma remained free from hemoglobin. Choroschilow believes that the red blood corpuscles of the cases of paroxysmal hemoglobinuria are easily injured by low temperatures, but hemolysis does not take place in the peripheral circulation and the injured red cells are not freed of their hemoglobin until they reach the kidney.

Polyarteritis Acuta Nodosa and Peri-arteritis Nodosa.—CARNEGIE DICKSON (*Jour. Path. and Bact.*, 1907, xii, 31) believes that under the name "peri-arteritis nodosa" two distinct diseases have been described; a true peri-arteritis, nodular in its distribution and usually due to syphilis, a condition for which he would restrict the term peri-arteritis nodosa, and a second disease in which all the coats of the vessels are affected and which is characterized by the formation upon the smaller and medium-sized arteries of small localized nodules, polyarteritis acuta nodosa. This latter condition is exceedingly rare and Dickson has only been able to find 18 reported cases. He describes in detail a case of this latter type occurring in a boy, aged fourteen years. The disease started with severe abdominal pain later the patient developed symptoms

suggesting meningitis, and, finally, died after an illness of ten weeks. At the autopsy small nodules were found upon the small vessels of the mesentery, heart, liver, spleen, kidneys, brain, and spinal cord. There was subacute nephritis with infractions of the kidneys and lungs, and hemorrhage from rupture of small aneurysms of vessels upon the surface of the brain. Histological examination of the nodular formations upon the arteries showed that they were in almost all cases small aneurysmal dilatations of the vessel wall, sometimes involving the entire circumference of the vessel, sometimes involving only one portion of the wall. The formation of these aneurysms could be followed in the smallest and earliest nodules. The first changes occurred in the muscular coat, and were of a degenerative and inflammatory nature; later the elastic lamina appeared broken and fragmented. Following upon this came the aneurysmal dilatation with proliferative changes in the intima, frequent thrombosis of the vessel lumen, and inflammatory changes in the outer coat. Giant cells were often seen near the fragmented elastic lamina. The etiology of the condition (which is very obscure) could not be elucidated by a study of this case. No bacteria, including the tubercle bacillus, could be found in the tissue, and the stains for Spirochæta pallida gave negative result. Dickson, nevertheless, believes that the condition is caused by some bacterial or other infective organism or its toxin. Since the vasa vasorum of the diseased vessels showed some alterations, Dickson thinks it possible that the infection reaches the vessel wall either through this route or by means of the perivascular lymphatics.

Action of Magnesium Salts on the Peripheral Motor Nervous System.—The interesting experiments which Meltzer and Auer have performed regarding the action of magnesium sulphate and the results which they have obtained following the subcutaneous and intraspinal injections of this salt, raise many questions regarding the physiological action of the drug upon the nervous system. BARBIER (*Jour. de Phys., et de Path. Gen.*, 1907, ix, 611) has made some experiments by means of the ergograph to determine what action the injection of magnesium salts has upon the nervous system of the frog. He finds that after such injections, the muscle responds to excitations of the nerves of the leg in an irregular manner, producing a curve which is typical, according to Mosso and others, of curves obtained with the use of poisons acting after the manner of curare. The magnesium salts affect only the motor endings of the nerves, for the muscle reacts perfectly to electrical stimuli. The ergographic curves show the progress of the intoxication which ends in complete paralysis of the motor nerve terminals.

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